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A.M.A. ARCHIVES OF OTOLARYNGOLOGY

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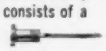

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Recurring Nasal Polyposis

A Rational Procedure for Its Prevention

A. R. HOLLENDER, M.D., Miami Beach, Fla.

The problem of recurring nasal polyposis continues to plague the rhinologist. It also continues to harrass the patient whose relief of nasal obstruction following repeated operations is all too brief. Excision of polyps usually produces no cure, even though in some instances resort is had to radical surgical procedures on the nasal sinuses. The large number of patients subjected to repeated polypectomies rationalizes the urgent need for improved management. The situation cannot be ascribed to the inadequacy of current surgical methods. The fault rests more on the fact that excision in itself fails to correct the factors favoring regrowth of hyperplastic tissue.

Efforts have been made in the past to classify nasal polyps according to their histologic contents. Such information is, of course, only of academic interest. Whether polyps are true neoplasms is still a matter of controversy. According to Ash,[1] neoplastic proliferation of connective tissue is at times a feature, "but ordinarily the polypoid character is the result of edema, chronic mononuclear and eosinophilic exudate, vascular dilatation, and at times, cysts. These are of two types: true retention cysts developing in occluded mucous-gland elements, and false cysts without a cellular lining, the result of pooling of the serous fluid. Secondary lymphatic follicles are not an uncommon finding."

Submitted for publication Sept. 24, 1957.

Professor of Otolaryngology, Emeritus, University of Illinois College of Medicine; Clinical Professor of Otorhinolaryngology, University of Miami School of Medicine; Consulting Otolaryngologist, Variety Children's Hospital, Miami, Fla., and Mount Sinai Hospital and St. Francis Hospital.

Review of Previous Studies

In one of my earlier articles,[2] I reported the results of clinical studies which dealt mainly with the prevention of recurrence of nasal polyps. For this purpose resort was had to the postoperative use of zinc ion transfer or radium. These supplemental nonspecific measures seemed warranted on a trial basis, though radium application had at times been advocated as a preventive measure.[3-5] In a subsequent article,[6] I compared the results of the postoperative use of zinc ion transfer and radium. I was led to make this comparison not because radium therapy had proved unsatisfactory, but because some rhinologists objected to it, and, moreover, in some quarters, radium was not easily available.

After 10 years, I again presented a report[7] of my experience with the methods previously described. At this time I outlined a definite plan, step by step, in the hope that others would put it to the test and place their results on record.

In still another communication,[8] I discussed the potential causes of polypoid recurrence. It was my conviction that some

of this information eventually would lead to a solution of the problem concerned.

Allergy in Relation to Nasal Polyps

If polyps have only an allergic genesis, there is lack of general concurrence of this concept. Despite the failure of allergic methods to prevent recurrence of polyps, the role of allergic factors cannot be completely disregarded. If polyps will disappear strictly under the influence of an allergic regimen, Harpman's [9] claim that this is unusual should be considered invalid. Because he has not experienced the favorable response claimed by some allergists, he has found it necessary to resort to operative intervention to effect a cure.

Hargrove [10] contended that polyposis is a symptom of a generalized disease whose cause is a sensitization to a particular antigen. In his opinion, it is doubtful that polypi can form from a chronic state of infection with no allergy present.

Hansel's [11] view concerning the formation of nasal polyps is the one now generally accepted. It is his belief that in most instances in which allergic reactions have occurred over a long period of time, chronic edematous thickenings and polyps eventually make their appearance.

According to Davison,[12] hyperplastic sinusitis is due to localization of the submucosal tissues to bacterial proteins, most often those of the Gram-positive cocci. He did not think that polyps are due to food or inhalant sensitivity.

Polyps probably originate as a productive inflammation, which in many instances is allergic in nature, in the opinion of Karsner,[13] who stated further: "In some instances glandular acini are numerous enough to suggest a true adenoma, and in others the mass may be angiomatous. Generally, however, they are merely masses of edematous connective tissue covered by columnar epithelium which may show metaplasia."

From the foregoing excerpts, it is difficult to refute the contention that allergy plays a dominant etiologic role in nasal polyps. It is not, however, the sole cause as argued by most allergists. There is ample evidence which points to an interplay of other causes.

Interplay of Causes

On the basis of clinical, allergic, and histological observations in 50 patients with nasal polyps, Clerici et al.[14] concluded that the etiopathogenesis of polypoid growths may be ascribed to four different factors: the anatomical structure of the rhinosinusal mucous membrane, the vasomotility of this mucous membrane, a constitutional condition of extra-allergic hyperergy, and an attenuated chronic inflammation. In the opinion of these workers, none of these factors could by itself explain the genesis of polyps, but all of them cooperate in their formation.

Recently the virus theory of nasal polyp etiology was advanced by Weille and Gohd.[15] In support of this theory, they stated: "The tendency toward slow or rapid recurrence of nasal and sinus polyps after excision, their spontaneous appearance and disappearance with the onset and subsidence of head colds in certain patients, and their slowness or absence of recurrence after diathermy needle coagulation of the postexcision stumps, united to suggest the possible viral etiology of these polyps. It was reported, in 1950, by Weille [16] that viral infection seems important in certain allergic states. If this observation is correct, then the hypothesis of a viral etiology for polyps merely elaborates upon the present widely accepted relationship in many cases between polyposis and a background of bacterial allergy."

In a previously published article I stressed that repeated removal of nasal polyps is a factor hitherto never considered as leading to regrowth of polypoid tissue. The trauma of frequently repeated operative interventions probably acts as a stimulus to regrowth of the tissue. Hargrove [10] pointed out that repeated traumatization of the cells leads to a progressive infective condition, and this will cause a proliferative osteitis. Uncontrolled infection or allergy, or both, is conducive to regeneration of hyperplastic tissue,

and with the activation incident to trauma from any source, this regeneration is hastened and emphasized.

Influence of Corticosteroids on Nasal Polyps

Introduction of the corticosteroids held out some hope for the nonsurgical elimination of nasal polyps. The results, however, have been disappointing. It is of interest, nevertheless, to review the effects of some of these agents on hyperplastic tissue.

Owen [17] injected hydrocortisone free alcohol (Cortef) into polypoid masses. Although he was able to reduce the size of the masses, and thus afford patients subjective improvement, he was unable by the injection technique to eliminate the polyps entirely.

In a study of 30 patients, Hotchkiss [18] adminstered predisone (Meticorten, Schering) according to an arbitrary and tapering-off schedule, until a small maintenance dose seemed adequate. In this series of patients, besides the nasal polyps, anosmia was present in most instances. After seven days of treatment, the shrinkage response was observed to be in direct proportion to the fluid content of the polyps. "In the case of mucoid polyps the response was dramatic . . . In the fibrous type of polyps, the shrinkage was negligible, or at least less pronounced than in the mucous polyps, and the return of the sense of smell was less dramatic."

Although Hotchkiss was aware that the newer hormonal preparations produce temporary disappearance of nasal polyps, he was impressed with the dramatic results following the use of prednisone. He advocated a maintenance average dose of 3 mg. per day for prolonged action.

One of Samter's [19] objections to the use of corticosteroids in pathologic nasal processes is that these processes frequently become unmanageable after administration of the hormones. "As compared with repeated polypectomies the severity of episodes of asthma in the elderly patient justifies preference for the use of corticosteroids as opposed to surgical treatment wherever possible."

Bordley [20] examined nasal polyps removed from a series of patients. He observed that the growths become opaque and pink and began to shrink, disappearing completely in many patients. More often, however, they returned in from two weeks to two months, but could be made to regress with nasal sprays of cortisone.

The studies reviewed and numerous others not included here render it clear that the corticosteroids have not fulfilled the permanent effects anticipated from them in eliminating nasal polyps. Moreover, there are definite contraindications to the use of corticosteroids, and these contraindications must be observed before this therapy is undertaken. There are, however, certain indications for which hormones represent constructive therapeutic agents, and as such, their contribution to present methods of dealing with nasal polyps must be appreciated. It should be emphasized again that this approach is purely of a palliative nature, and though it serves a valuable purpose at times, it has not replaced the conventional procedure of excision.

Plan of Effective Control of Recurring Polyposis

Though surgical extirpation is the method of choice for elimination of nasal polyps, in itself it has proved inadequate as a cure. This applies with equal force to radical operations on the nasal sinuses. The absolute necessity for some form of supplemental therapy cannot be disregarded in the light of present knowledge concerning the frequent incidence of polypoid recurrence. Often the need for allergy control is obvious. This may entail a change of environment or occupation, and sometimes habits of living, but usually the situation, if it exists, is more complicated. A methodical study of the patient may have to be done for diagnostic purposes. If the nose is massively obstructed, however, time is the factor which may guide the rhinologist to postpone all

supplementary procedures until the nose has been cleared of the polypoid growths.

To prove effective the operative program should begin with the preparation of the patient. As a preliminary step the nose and its accessory sinuses should be freed of infection. If resistance is encountered, resort should be had to sensitivity tests to avoid guesswork in prescribing antimicrobial agents. If in addition local measures are required to clear the nasal or sinus infection, indicated procedures should be instituted at the opportune time.

After the nose has been rendered reasonably free from infection, operation is undertaken. It is assumed, of course, that the patient has been hospitalized and that the polypectomy will be performed in the hospital operating room. This is, despite arguments to the contrary, a major procedure and should not under any circumstances be performed in the office. The secret of success is the accomplishment of meticulous removal of all visible polypoid tissue, then either curetting the bases of the polyps, as advocated by Harpman,[9] or electrodesiccating them.

After completion of the operation, the rhinologist has his choice of applying radium immediately, or waiting until the healing process has subsided. Findlay[21] employed the former plan in 36 of a series of 40 patients without harmful effects. In four of the patients there was an interval of 7 to 28 days between operation and insertion of radium. My own preference is to await intranasal healing before the radium is applied. As a general rule, the interval is two weeks. During this period the utmost in intranasal hygiene should be maintained.

Frequent intranasal inspections are imperative to ensure the rhinologist that residual tags have not made their appearance. If some present themselves, they should be electrodesiccated promptly. Usually, a small amount of topical anesthesia suffices for the purpose. At each inspection, an intensive search is made for recurrent tissue, which is destroyed electrosurgically almost as quickly as it presents itself. In this way no time is allowed for regrowth of polyps.[22]

The irradiation procedure has been outlined in detail in previous papers.[7,8] It consists of applying a 50 mg. capsule of radium, correctly screened and encased in gutta-percha. The capsule is placed against the area of operation in the nasal cavity for a total period of three hours. Occasionally, two areas should be treated, in which case the time is divided and the applicator moved from one area to the other. Each nasal chamber is treated separately, preferably on different days, although both chambers may be treated simultaneously if two capsules of radium of 25 mg. each are available. As a rule, two or three applications are necessary with intervals of three weeks.

A word of caution is in order concerning the handling of radium applicators. It is assumed that the rhinologist will familiarize himself with these facts before undertaking radium therapy.

Causes of Failure in Preventing Regrowth of Nasal Polyps

In a study of this problem extending over a period of more than 20 years, I have constantly sought for the causes of recurrent polyposis. I have concluded that the major cause of failure can be laid to the indifferent attitude of the rhinologist in pursuing a methodical plan of treatment. The piecemeal office technique has been proved to be highly unsatisfactory. Even in patients in whom the allergic factor has been adequately controlled preoperatively, elimination of infection and meticulous excision, as can be performed only in the hospital, are highly important requisites.

Radical operative procedures on the sinuses are no guarantee that polypoid regrowth will be prevented. It is clearly evident from the experience of Harpman[9] and others, that something more than excision is necessary for prevention of recurrent polyposis. Although the efficacy of allergy control is not disputed, this means has more theoretic than practical significance. In the light of experience and present

knowledge, though probably not the final answer, postoperative irradiation promises the most satisfactory means of preventing recurrence of nasal polyps.

The situation with polypoid growths may be likened to that of the adenoids, whose total removal, even under direct vision, is well nigh impossible. It is not a matter of operative technique or skill of the surgeon. Regrowth of adenoids has occurred even after the most meticulous extirpation. The difficulty lies in the fact that a type of tissue is being dealt with which regenerates rapidly to form grossly invisible shreds. The trauma of the operation only adds stimulation to the regenerative process. With nasal polyps, one is confronted with an analgous problem.

I have just mentioned that operative trauma stimulates the regenerative process of certain tissues. Obviously, with nasal polyps, frequent removals lead to a cumulative effect which definitely enhances the regrowth of these hyperplastic tissue masses.

Comment and Conclusions

Despite the positive claim of allergists that polypoid recurrence can be prevented by an allergic regimen, such a development has proved to be the exception and not the rule. The problem of nasal polyps is elementary, but its ramifications are numerous, and at times complicated. Resort to nonspecific procedures to prevent recurrence of nasal polyps is not the ideal. This approach is, however, the most effective means now available.

Corticosteroids as a palliative represent a constructive therapeutic contribution. Because of failure to produce permanent effects, and their established contraindications, they possess only restricted therapeutic value. The hazards of prolonged administration of the hormones suggest the need for caution, a fact mitigating against dependence on this type of therapy.

Recurring polyposis is preventable if strict attention will be paid to the planned procedure previously outlined. The necessity of classifying polyposis as a major problem to be dealt with only in the hospital operating room cannot be sufficiently emphasized. Meticulous excision and postoperative radium irradiation promise highly satisfactory results.

In a series of 250 patients during the past 23 years, follow-up reports from the large majority of the patients treated disclosed favorable results in approximately 75%. This high percentage of successes has warranted the detailed care provided in the planned procedure here advocated.

Summary

Recurrent nasal polyposis represents a problem which continues to plague the rhinologist.

A review of past experience suggests that the problem is not beyond control by available methods.

Despite the failure of allergic management, the role of allergy in nasal polyposis cannot be disregarded.

Sufficient attention has not been paid to the interplay of several factors in the etiopathogenesis of nasal polyps.

The influence of corticosteroids on nasal polyposis is strictly palliative, but as such they provide the rhinologist with an effective weapon in dealing temporarily with critical situations.

A definite plan for the control of recurrence of nasal polyposis is recommended.

Meticulous excision of the polypoid growths, as practiced in the hospital, and postoperative radium irradiation promise a high percentage of successful results in preventing recurrence.

Causes of failure include various factors, the more important of which include an indifferent attitude of the rhinologist in following a methodical plan, resort to the office piecemeal technique, failure to cure infection before excision is attempted, and dependence solely on operative extirpation.

605 Lincoln Rd.

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External Nasal Pyramid and Septum Reconstruction

Report of a Case

LEWIS W. JORDAN, M.D., Portland, Ore.

This record depicts the steps performed in a nasal reconstruction based on functional concepts. The operation was performed on July 20, 1956, in the Memorial Hospital of Cheyenne, Wyo., by Ralph H. Riggs, M.D., assisted by Russell I. Williams, M.D. The report was written by me, who was observing from the surgery balcony about 15 ft. from the operative field and viewing through a binocular field glass. The procedure was a group demonstration included in the program of the American Rhinologic Society.

The purpose of this presentation is to illustrate the techniques of operation and a method of clinical teaching which have long been used by Maurice H. Cottle,[1] M.D., of Chicago. It also shows what an observer with training can see and think from a distance. It emphasizes the value of writing the record while the operation is in progress, so that all details and comments can be vividly and accurately included. Each observer can and does write his own record as a means of "learning by doing."

Above all, the purpose is to emphasize those parts of the surgical technique which make possible the reconstruction of both the nasal septum and the external pyramid in a single operative procedure.

Report of Case

Description of Patient.—The patient was a laboring man, 51 years of age, who had a large and long nose with the distal portion severely twisted to the right. His facial skin was heavy, course, and tanned. These features, combined with his apparent satisfaction with his social and economic fate in life, obviously did not entitle him to expect the attainment of the facial features possessed by a movie actor.

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He stated that he sustained a nasal injury at about the age of 6 years. For many years his nose had been obstructed; a postnasal discharge had been present, and he had had periodic headaches. He had not responded to the usual treatments for such hypothetical conditions as "sinus disease" and other popular syndromes.

Detailed examination showed that the distal three-quarters of the entire nose was deviated severely to the right, the deviation increasing as the tip was approached. The proximal 1 cm. of the nasal bones was in the midline. Below this, the bones began to deviate to the right, which probably indicated the site of the old fracture. The nasal bones were fairly long, and the cartilaginous vault was short. The short side of the pyramid was on the right and the long side on the left. The pyriform aperture was 4 mm. higher on the left than on the right. There was considerable scar tissue subcutaneously on the left side of the nasal tip. The base of the nose was broad, and the septal concavity was on the right. The septum was severely impacted on the left (in contact with the left middle and inferior turbinates), while marked ballooning was present on the right and the valve was closed on the left. (The nasal valve is the angle between the cartilaginous septum and the upper lateral cartilage. The flow of air through the nose is regulated by this structure. When it is excessively wide, it is said to be "ballooned.") The right dome was larger than the left, and the tip was cleft. The left lobular cartilage receded and was more cephalic than the right. It appeared that returning was present on both upper lateral cartilages. ("Returning" refers to a cephalic external angulation of the distal edge of the upper lateral cartilage. This deformity stiffens the cartilage and limits its flexibility.) The nasal index measurements[2] were 28/55 which gave an index of 50. This indicated a Mesorrhine type of skull. The tip measurements of 30/44 gave an index of 68. (The patient should be told that occasionally more than one operation is required for a complete correction.) A complete detailed record of the examination and procedure was written as the operation progressed.

Fig. 1.—Location of injections for local anesthesia of nose. (1) External nasal nerve; (2) infratrochlear nerve; (3) infraorbital nerve; (4) nasopalatine nerve.

Medication and Anesthesia.—The preoperative medication consisted of promethazine (Phenergan) hydrochloride, 50 mg. and barbital (Veronal), 10 grains (0.6 gm.) by mouth one hour before surgery; chlorpromazine (Thorazine), 25 mg. orally one-half hour before, and meperidine (Demerol), 100 mg. intramuscularly at the time of surgery. As the patient was prepared, the earphones were adjusted so that he heard the music properly. (Music during surgery performed under local anesthesia has a very calming effect.) After the vibrissae were cut, cocaine crystals in epinephrine (Adrenalin) 1:1000 were applied topically over the sphenopalatine foramen area and the nasociliary nerve. At the same time lidocaine (Xylocaine), 2% with epinephrine 1:100,000, was injected into the infratrochlear, external nasal, infraorbital, and nasopalatine nerve areas bilaterally. The needle was inserted at the alar facial junction bilaterally (Fig. 1). A total of about 9 cc. was used.

Septal Surgery.—A vertical incision was made in the right membranous septum (Fig. 2*A*) and elevation of the perichondrium on both sides of the distal portion of the septum was begun (Fig. 2*B*). This was continued on to the complete elevation of the right septal flap. With the Stevens scissors, the base of the nose and the upper lip were undermined (Fig. 2*C*). As a continuation of the same maneuver, the nasal spine was exposed. Elevation of the left septal flap was next begun. The separation of the septal cartilage from the nasal spine released the tension, and the septal cartilage moved to a position near the midline. As a result, the tip was carried into a more normal position. (This clearly illustrated one of the close interrelations between the septum and the external nose. The pathology of these two structures was so closely related that one could not be corrected without attention to the other. It demonstrated the fallacy of attempting to disregard one of these structures in the correction of the other.)

For the present, the distal end of the septum was retained. Elevation of the left septal flap was continued, and the deviated portions of the septal cartilage and bone were removed in small fragments. The cartilage was found to be almost ossified. The anterior nasal spine was further exposed with the Cottle knife. (This versatile instrument is invaluable in such areas as this.) The spine was mobilized with the 4 mm. chisel and rongeur. Removal of the spine in this case was not indicated because of the possibility of producing retraction of the labiocolumellar angle. (Removal might also lead to a later broadening of the nasal base.) By this time most of the septal deformity had been removed, with the possibility remaining of the removal of more septum later if required. A dry gauze packing was inserted in both sides of the nose.

Lobular Cartilage Surgery.—Further examination showed that the right nasal bone was shorter than the left. Intraseptal separation of both nasal bones via the septal space was carried out. In doing this, the 12 mm. chisel was inserted above the

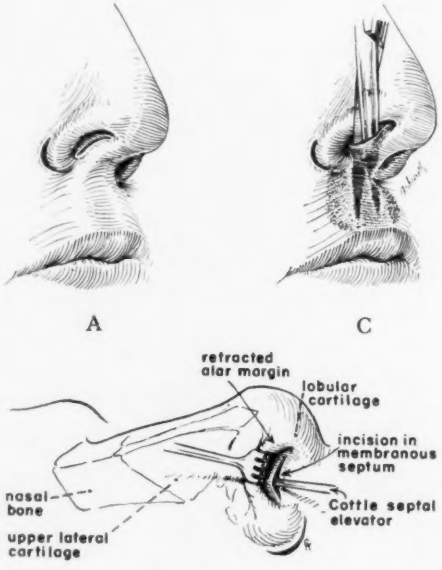

Fig. 2.—*A*, incision in membranous septum. *B*, elevation of right septal flap. *C*, undermining of the base of the nose and the upper lip.

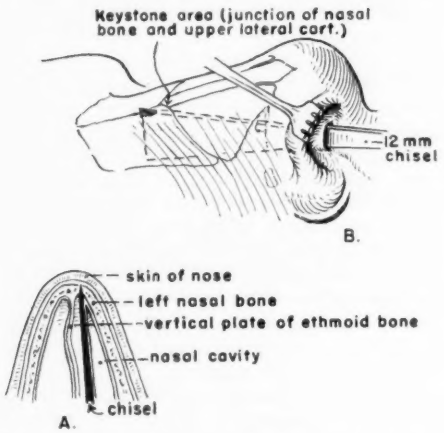

Fig. 3.—*A*, insertion of 12 mm. chisel beneath periosteum of left side of septum for separation of nasal bone. *B*, insertion of chisel above keystone area to preserve the continuity of the nasal roof.

keystone area so as to preserve the continuity of the roof (Fig. 3). (The keystone area is the junction on the septum of the nasal bones and the upper lateral cartilages.) This was possible since no hump removal was necessary. The right dome was found to be higher than the left, and the tip was cleft. The left lobular cartilage receded and was more cephalic than the right. A guide suture was inserted at the point where the lateral flare of the medial crurae began. There was much scarring and thickening in the left dome. Bilateral slot incisions were made just distal to the edge of the lobular cartilage (Fig. 4). (This movement must be very precise so as not to damage the distal edge of the cartilage. It is surprising to note the high proportion of new disposable knife blades which are too dull for this work.) The lobular cartilages were exposed with the Stevens scissors, and the skin elevation was

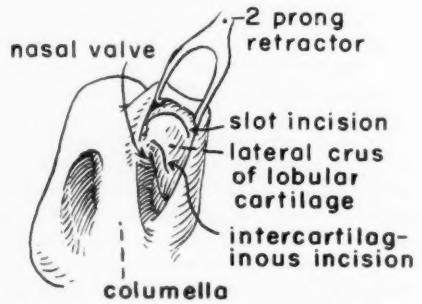

Fig. 5.—Intercartilaginous incision and slot incision.

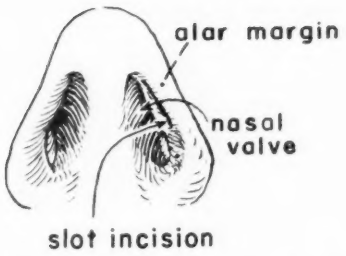

Fig. 4.—Location of slot incision distal to the lower edge of the lobular cartilage.

continued up over the nasal dorsum. The left intercartilaginous incision (Fig. 5) was somewhat difficult owing to scarring in the cul-de-sac. The right incision was simpler. Through these incisions, extensive elevation of the dorsal skin was completed with the Stevens scissors (Fig. 6). (Use of the scissors instead of the knife for this purpose is simpler and safer.) The septal pocket was also enlarged with the scissors. The right lobular cartilage was cut somewhat lateral to the dome. A similar incision was made in the left dome in a more lateral position to compensate for the smaller cartilage on this side. The latter procedure was again more difficult than the right because of the scarring. Both sides were cut on the Neivert hook. (This method requires a more extensive skin elevation than with the use of the lower lateral clamp.)

The organ of expression was removed on the right side. (Removal of this muscular organ decreases the circulation to the subcutaneous tissue and thereby gradually causes a thinning of the skin. This is of advantage to a patient, such as this one, who has a thick skin.) The right lateral crus was shortened about 5 mm. (In shortening a long nose, several structures, including the lateral crura, must be shortened.) The tip of the lateral crus was bared 4 mm. This allowed the lateral crus to overlap somewhat the medial crus in the narrowing of the tip. The small area of freed skin also helped to line the dome. The scarred

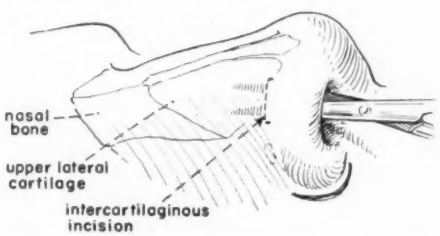

Fig. 6.—Elevation of dorsal skin with Stevens scissors inserted through intercartilaginous incision.

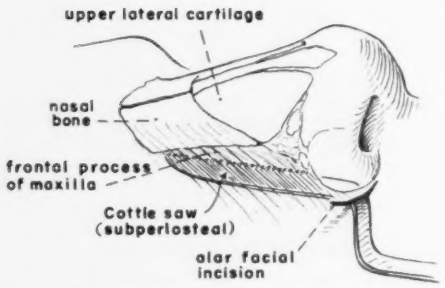

Fig. 7.—Osteotomy saw inserted through alar facial incision.

organ of expression was removed from the left side. A returning of the cephalic margin of the left lobular cartilage was found. This area was shortened 3 mm. Scar tissue was removed from the subcutaneous area of the left side of the tip. The tip of the left lateral crus was bared 3 mm. For the purpose of narrowing the tip, a small amount of areolar tissue was removed from between the medial crura. Two small vertical grids were made in the right medial crus because the right dome was larger than the left. The medial crura were sutured together to narrow the tip and to eliminate the cleft.

Nasal Bone Surgery.—Base lines were drawn along the naso-optic groove, and the nasal bones were outlined with methylene blue. Alar facial incisions 6 mm. in length were made bilaterally (Fig. 7). The osteotomy was carried out with the special Cottle saw. (The hack-saw teeth on this instrument cut faster than the usual bone saw and also pull the bone dust out of the wound.) Since the nose was deviated to the right, the left nasal bone was the broader of the two and formed the longer side. The cut on the right side was made at the level of the base line. On the left side the cut was made above the base line so as to make both sides of the nasal pyramid equal. The bone dust was raked out. The osteotomy was completed with a narrow straight chisel and the curved 7 mm. chisel. The nasal bones were infractured and the frontal spine was fractured. It was now found that the heavy bone of the vomer overlapped the left side of the ethmoid plate. Part of the latter structure was removed to correct the overlapping.

Upper Lateral Cartilage and Other Surgery.—The right upper lateral cartilage was exposed and found to be short with moderate returning. Freeing of this structure partially relieved the returning and the consequent ballooning. (Tension in the subcutaneous tissues frequently causes returning, which in turn can cause ballooning.) Subcutaneous scar tissue in this area was removed, but the cartilage was not shortened. The left upper lateral cartilage was exposed and found to have much returning. In fact, there was a double returning to form an S. This was probably a result of the severe scarring in the left side of the nose. The cartilage was shortened 3 mm. More maxillary crest was removed, and the permanent cod liver oil packing was inserted after the original temporary gauze packing had been removed. (This oil seems to be more compatible with nasal secretions than petrolatum and does not produce such a disagreeable odor. The packing is applied loosely in layers and holds the nasal membranes and solid structures in the proper position.)

At this time the distal end of the septal cartilage was attached only ventrally and could be moved into the proper midline position. (This structure with its normal perichondrial attachment is less likely to be absorbed and to produce a retracted columella than would an implant, autogenous or isogenous.) To produce a more solid septum, four pieces of the patient's septal bone and cartilage were replaced in the septal space. They were maintained in position by the packing. Three septocolumellar sutures of 000 plain suture material were inserted to close the membranous incision. One figure-of-eight suture of 00 plain suture material was inserted in the base of the columella. A piece of autogenous septal bone was inserted over the spine area. Chromic base sutures of 00 size tied over autogenous septal bone buttons were inserted (Fig. 8). (This procedure, combined with proper reconstruction of the columella tends to narrow the base of the nose and to prevent later depression of the tip and sagging of the columella. This deformity is caused by a combination of scar tissue contraction and gravity. The contraction of the scar tissue in the base of the nose is a powerful force.) The alarfacial incisions were closed by 5-0 Dermalon sutures (skin sutures processed from nylon) on each side, inserted by small plastic surgery needles. The lateral crus of the left lobular cartilage was cross hatched to correct a concavity. Submucosally, the left upper lateral cartilage was separated from the septum 1.5 cm. and the right 2.0 cm. The cephalic portion of these cartilages was not separated in order to preserve the keystone area. The septum was not completely removed. Eighteen autogenous septal fragments were inserted over the upper lateral cartilage area, more being inserted on the left under the original depression. (These flakes may eventually tend to work cephalically and to be absorbed. However, in the meantime, they help to form a firm and smooth fibrous support of the subcutaneous tissues.) At the completion of the surgery, the patient was shown his postoperative picture. (This assures him that his deformity has been corrected. He is, therefore, more willing

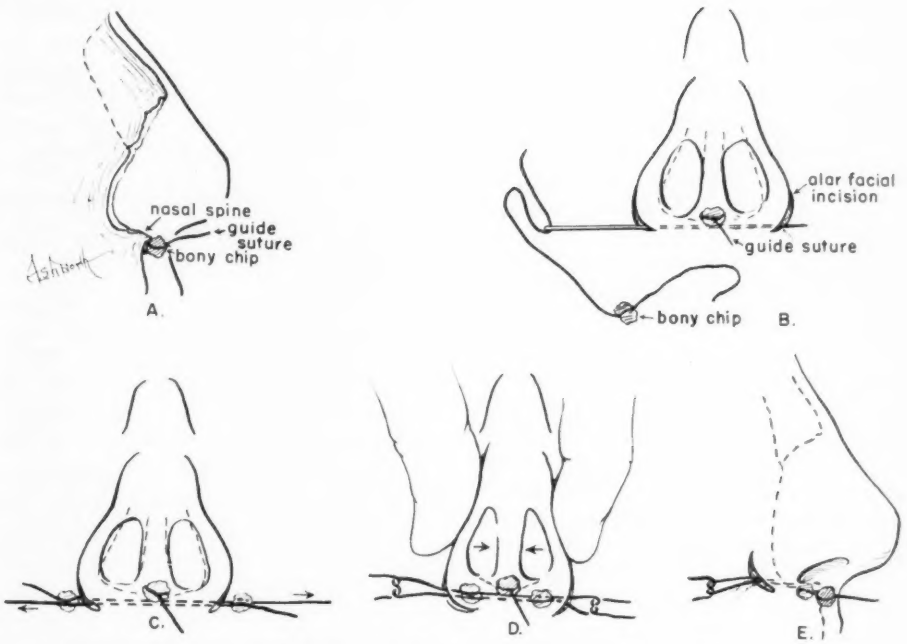

Fig. 8.—Cottle reconstruction of nasal spine area. *A*, insertion of bone fragment over spine area. This is inserted through septal incision with a guide suture. *B*, insertion of base suture through alar facial incision. *C*, insertion of base sutures. *D*, tying of base sutures over bone buttons to narrow the base of the nose. *E*, final position of bone implants.

to tolerate the external dressing and the eye bandages.)

External Dressing.—In the application of the external dressing, care was taken to have the adhesive smooth and with no wrinkles. The entire nose, including the tip, was covered with adhesive. Next, the stent splint was applied. (The application of the external dressing is one of the most important parts of the entire operation. The adhesive helps to keep the skin modeled and to give it a semirigid support. The covering of the tip helps to mold its contour and to prevent edema. If several layers of tape are applied, they act almost as a rigid splint. The rigid stent splint has several functions. It should not be applied with the idea of holding a deviated nose straight or with the purpose of bending a bony fragment into place when this fragment has not already been completely mobilized. When properly used, the rigid dressing should be literally poured on after the edema and hemorrhage have been gently massaged out and the various components of the nose have fallen into their new natural positions. It is not applied with the idea of forcing any structure into an unnatural position or into a position of tension. In other words, after the nose is in good position and size, the softened stent is draped over the nose to retain the position. The stent is hardened at once with ice packs. The rigid dressing protects the nose from external damage, and its pressure, which does not have to be very great, prevents and decreases edema. An unsplinted nose would swell tremendously. Gentle pressure with pads on the eyelids tends to prevent and decrease edema. Since the eyelids may become swollen, the patient could not see very well even though the eyes were not covered. Occasional patients, especially those who are nauseated, are very apprehensive because of the eye patches. In these situations, the eyes should be left uncovered.)

Rationale of Procedure

In considering this rhinoplasty and septal reconstruction in retrospect, a very important consideration is the fact that every structure in the nose was exposed and put into its normal position. From this standpoint, the entire operation might be considered radical. On the other hand, very little tissue was actually removed, and the small amount which was, was replaced as a part

of the reorganization. In other words, displaced parts were replaced into their normal position. In this respect, the procedure was very conservative. Since the dissection was all very meticulously done there should be a minimum of postoperative scarring. Gentleness in the handling of tissues prevents devitalizing and thus infection. If secondary surgery is necessary, it can be carried out rather easily. Every precaution was taken to prevent damage to structures. This was illustrated by the use of the ala protector, very sharp knife blades, attention to proper planes of separation, guarding of sharp instruments, etc. An extremely important consideration is the preservation of all mucous membranes and skin. If these remain intact, a minimum of fibrosis results, and the form of the nose can usually be preserved. In the event of an inadvertent excessive loosening of a nasal bone, packing can be inserted under the mucosa to maintain its position. A minimum of short incisions also tends to prevent scarring.

Effect of Nasal Reconstruction upon the Patient

Psychologically, a rhinoplasty is very important to the patient. Great care must be taken preoperatively to determine the real motive of the patient. The essential parts of the operation should be explained to him before surgery. During the procedure he must know that everything is being done for his best interest. After the operation, especially at the first dressing, his newly corrected nose must be presented to him in a tactful manner. Any uncomplimentary remarks at this time may completely upset his sense of satisfaction. He must be assured that it will take considerable time for the edema to subside completely and for the final result to be attained. When this desired result has been achieved it is remarkable to see the great improvement in the nasal function as well as in the cosmetic appearance. The patient often steps forth into a better life of new attainments.

Summary and Conclusions

A detailed description of the reconstruction of the nasal septum and the external nasal pyramid in a single combined operation according to the technique of Cottle et al. is presented. Methods of combining the various steps into a single safe procedure are illustrated. This is in contrast to the two-stage method often used, in which the septum is corrected in a separate operation.

A method of clinical teaching is presented, wherein an observer sitting in a surgery balcony can actively participate in the operation. This is accomplished by the use of binocular field glasses and the making of detailed notes and comments as the operation progresses.

The contribution made by the patient in such a teaching program must never be forgotten. His voluntary participation in the plan makes practical teaching possible and also partially compensates for the functional benefit which he receives.

624 Medical Arts Building (5).

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Rhinoplasty

A Simplified Technique

SAMUEL CLINE, M.D., Boston

During the past 15 years there has been a steadily increasing interest on the part of otolaryngologists in the technique of rhinoplasty. This has been manifested by the many papers published on the subject. The reason for this interest is undoubtedly the realization that there are available techniques which can not only serve a cosmetic and psychologic purpose but can also correct faulty function.

Many papers have concentrated on a single aspect of the procedure. Others have been either too verbose or too academic, and this has led to confusion. Some papers advocated procedures which were mechanically and physiologically unsound; such procedures were bound to yield poor results.

After several years of research into the currently used techniques, comparisons, variations, and revisions of details, a technique was evolved which is simple and gives very satisfactory results—both cosmetically and functionally.

The first step in planning any surgical procedure should be a complete evaluation of the patient and his problem. This includes history and complete physical examination. The immediate problem must be defined as to pathology, deformity, and ultimate goal of treatment. Basic prerequisites for the success of any rhinoplasty procedure are three:

1. Preoperative medication. The patient should enter the operating room completely relaxed and cooperative. The folly of attempting surgery on an apprehensive patient is obvious.
2. Proper visualization of the operative field. There is no short cut, no neat headlamp that can compare with the overhead operating-room light (properly deflected), supplemented by smaller floor-stand lights at either side of the table. This arrangement of lights gives adequate intensity in the various directions, without interference of shadows.
3. Anesthesia and hemostasis. Intranasal packing and liberal infiltration of the nasal dorsum will result in a dry and painless field. The all-too-common attitude of pride in using very little anesthetic material will often come back to haunt the operator during the procedure. The benefit to both the operator and the patient derived from complete relaxation and freedom from pain and excessive bleeding will more than justify an adequate preparation.

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Teaching Consultant in Otolaryngology, Boston City Hospital; Associate Member in Otolaryngology, Beth Israel Hospital; Member, Alumni Staff, Massachusetts Eye and Ear Infirmary.

Procedures

Reduction of Dorsal Hump and Shortening of Nose.—The usual intercartilaginous incisions are made, and then the skin over the dorsum is freely elevated and the septum transfixed. The tendency has been to remove too large a hump. I prefer minimal removal; rasps may be then used to further lower the bony dorsum.

The upper lateral cartilages should be separated in their entirety from the septum, and the intercartilaginous incision extended laterally so that the cartilages are freely movable. They then can be properly trimmed medially to the level of the new dorsum; excess tissue between the outer skin and the upper lateral cartilages can be removed.

The cartilaginous dorsum is lowered, and the caudal end of the septum is shortened. Rather than remove a triangle, I prefer to use a Bard-Parker blade No. 15 and trim the caudal end of the septum with fine shavings to the proper level of shortening.

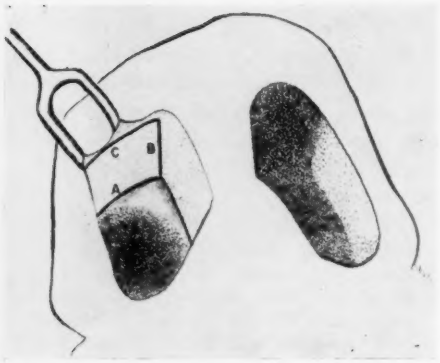

Fig. 1.—*A*, intercartilaginous incision. *B*, cartilage-splitting in incision. *C*, marginal incision.

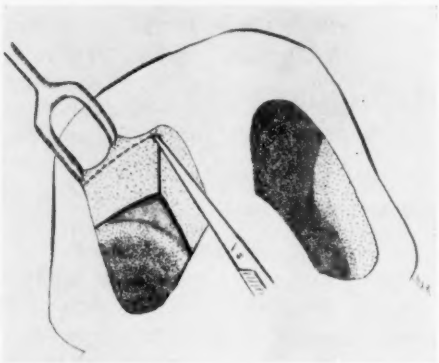

Fig. 2.—Separation with scissors of lateral crus at marginal incision from outer skin.

Submucous Resection of the Nasal Septum.—If a submucous resection is indicated, I prefer to do it at this stage, through the transfixion incision, leaving a strip of cartilage caudally. Only the parts of the septum definitely causing the obstruction are removed.

Before the tip is modeled, the septum is firmly united to the columella with several black silk sutures. All free edges of mucous membrane are carefully trimmed. It remains a matter of judgment, whether the septum should be invaginated into the columella.

In modeling the tip, I prefer the cartilage-splitting incision originated by Dr. J. Safian of New York and further modified by Dr. G. B. Fred [1] of Boston. This dissection allows freely moving lateral crura which can be trimmed inferiorly, superiorly, and mesially. Any underlying excess of tissue under the skin or between the mesial crura can also be removed. The upper borders of the mesial crura may also be trimmed and then united if necessary.

I have found that the end-result of this technique is a symmetrical and pleasing tip.

The accompanying semidiagrammatic drawings, are self-explanatory and illustrate the modeling of the lower lateral cartilages.

The cartilage-splitting incision divides the lower lateral (alar) cartilage into two parts, the lateral crus and the mesial crus, as in Figure 1. After the lateral crus is separated from the overlying skin it should be freely movable so that it can be displaced downward and outward without tension (Fig. 3). The lateral crus is then replaced, and the

Fig. 3.—Lateral crus pulled downward and outward. Outline of sections to be removed from lateral crus.

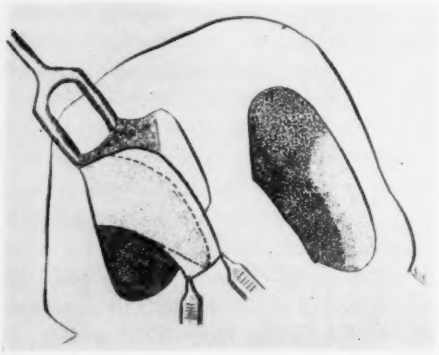

Fig. 4.—Lateral crus replaced and protruding triangle removed from inferior and mesial border.

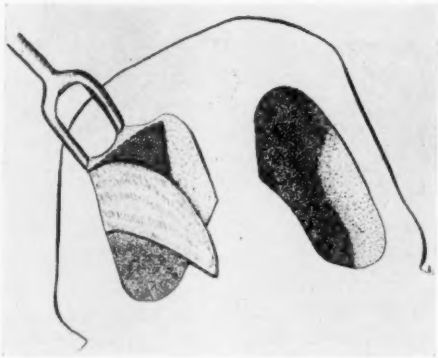

Fig. 5.—Appearance of lateral crus modeled.

protruding triangle is removed as in Figure 4; then the lateral cartilage is pulled downward and further trimmed mesially and superiorly as outlined in Figure 3. The final appearance of the lateral cartilage is seen in Figure 5. It is replaced in situ and sutured to the margin of the nostril, care being taken to have all raw edges covered.

It must again be emphasized that throughout the operation hemostasis is essential for a smooth technique. The use of gauze wipes, either dry or soaked with epinephrine, and further supplemented with suction are helpful.

The lateral osteotomy is performed intranasally. Saws may be used, supplemented by the chisel laterally and also between the septum and nasal bones. This step facilitates the outfracturing of the nasal bones, which can then be pressed together into the median line. Any roughness of the dorsum may then be filed with the rasp.

If inspection at the end of the operation shows the nostrils to be flaring and too wide in proportion to the lips, sections may be removed at the base of the nostrils. It is important that the sections be equal in size and position so that there be no subsequent asymmetry.

Summary

A technique for rhinoplasty and rhinoplasty combined with submucous resection that has proved uniformly successful has been described. Strict attention to details such as preoperative medication, visualization, and anesthesia of the operative field are essential. Correct analysis of the deformity and pathology of the structures involved, plus gentleness in the handling of tissue are rewarded by a pleasing result. The mysteries of rhinoplasty have been eliminated; the operation can and should be done by the general otolaryngologist.

416 Marlborough St. (15)

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Postoperative Nasal Hemorrhage

Report of Case with Pseudohemophilia

SIDNEY H. GIDOLL, M.D., San Francisco

The diagnosis and treatment of a hemorrhagic disorder may be relatively simple when the patient observed presents a pertinent history with discernible and corroborative physical and laboratory evidence. However, when there is neither history nor laboratory evidence to associate a profuse and persistent postoperative nasal hemorrhage with a blood dyscrasia, then the temerity of the observer to indulge in retrospection may perhaps be understood.

It is my purpose first to present a case report in which this challenging situation arose and then to follow this with a discussion of the clinical course, diagnosis, and treatment.

Report of Case

A 27-year-old male Puerto Rican was first seen in the office, Dec. 19, 1955, with the complaint of blockage of nasal breathing for the past two years. There was a history of headaches, frequent head colds, nasal discharge, constant sneezing, aching and watering of the eyes, and the occasional occurrence of hives. There was no history of abnormal bleeding or previous operations.

The nasal examination revealed the septum to be markedly deviated to the left, with a vomeric spur touching the left inferior turbinate. A compensatory posterosuperior deflection to the right was present. Both inferior turbinates were engorged with some mucosal injection.

Blood pressure was 120/80, and all other findings were within normal limits.

On Jan. 19, 1956, at 10:30 a. m. a submucous resection of the nasal septum was performed at Hahnemann Hospital. Preoperative and subsequent laboratory data are given in Table 1.

Medication preoperatively consisted of pentobarbital (Nembutal) 1½ grains (110 mg.) orally one hour prior to surgery and morphine sulfate 1/6 grain (10 mg.) and scopolamine hydrobromide, 1/200 grain (0.3 mg.) hypodermically one-half hour prior to surgery.

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Anesthesia consisted of equal parts of 10% cocaine and 1:1000 epinephrine chloride topically and 1% lidocaine (Xylocaine) 1:100,000 epinephrine by infiltration.

There was only minimal bleeding during the surgical procedure and, on completion, Simpson splints were tightly inserted on each side of the nose as packing. There was no bleeding whatsoever on leaving Surgery.

At 1:45 p. m., about two and one-half hours after his return from Surgery, patient began to bleed from both nostrils and this continued more or less profusely and intermittently for the next 19 days. That afternoon, about 5:30 p. m., the nasal packing was reinforced and empiric medication (such as estrone [Theelin], menadione [Hykinone], and Koagamin [a hemostatic preparation of oxalic and malonic acids]) was administered parenterally for bleeding.

The next day, Jan. 20, oozing of blood continued and erythromycin (Ilotycin) was given because of a slight fever.

The following day, Jan. 21, the nasal hemorrhage resumed more actively and soaked through the tight nasal packing. Since this was more bleeding than usually followed a simple septal resection, the decision was reached to explore the operative site in Surgery in order to locate the bleeding points.

Accordingly, 72 mg. of menadione was empirically given I. V., and under N_2O and thiopental sodium (sodium Pentothal) anesthesia, the old packing was completely removed from both sides of the nose. A postnasal pack was inserted and applied firmly against the choanae.

The old septal incision was reopened, and the suction tip placed in the space between the mucosal flaps. Active bleeding points on the vomeric ridge and the anterior nasal spine were controlled by touching with the Bovy coagulator. However, blood seemed to well up from the posterior portion of the septal border. Absorbable gelatin sponge (Gelfoam) and dry gauze packing were placed firmly in the septal space between the mucosal flaps, with the exit of the packing through the operative incision followed by 1 in. wide gauze packing in both nasal chambers along the entire depth.

At this time, surgical consultation was held with Dr. M. J. Groper because the possibility of liga-

TABLE 1.—*Laboratory Data in Case of Postoperative Nasal Hemorrhage*

Date	Bleeding Time (Duke), Min.	Clotting Time (Lee-White), Min.	R. B. C., Millions	HGB, Gm.	W. B. C.	Differential	Miscellaneous
1/19/56 Preop.	1½	4½	5.0	14.6	7,000	PMN 58 Lymph 37 Mono's 1 Eosin 2	Urine negative; serology nonreactive
1/20/56 Postop.	1	6½	5.0	15.0	7,800		Blood type A, Rh positive
1/21/56	8*	9½*	4.7	14.6	7,300	PMN 73 Lymph 24 Mono's 3	
1/22/56	6½	9½	4.7	15.0	8,700	PMN 73 Lymph 26 Mono's 1	P. C. V. 46%; Platelets 259,000
1/29/56	1	8½	3.9	11.2	15,000	PMN 82 Lymph 11 Mono's 7	P. C. V. 36%; Prothrombin time 80% normal
2/1/56			3.25	10.4			P. C. V. 30%
2/6/56			3.45	9.8	9,600	PMN 81 Lymph 14 Mono's 3 Eosin 2	P. C. V. 33%
2/20/56 †	2	3	4.0	11.8	8,200	PMN 78 Lymph 20 Mono's 1 Eosin 1	Capillary fragility neg.; tourniquet test; platelets 287,000; simplastin activity curve 96%
3/7/56 †				13.8			
5/2/56 1st Reentry	3	3½	5.2	15.6	11,800	PMN 37 Lymph 57 Eosin 6	Platelets 260,000; clot retraction complete in 1 hour
5/20/56 2d Reentry			4.7	15.0	7,200	PMN 69 Lymph 28 Eosin 3	P. C. V. 46.5%
5/25/56 3d Reentry			4.7	15.6	10,400	PMN 65 Lymph 34	Fibrinogen 250 mg/100 cc.; total protein 6.1 gm/100 cc.
5/31/56 3d Reentry			4.25	11.8	6,300	PMN 81 Lymph 19	

* At this time these abnormal readings were felt to be due to the presence of active bleeding and within the extreme range of laboratory techniques for normal.
† From Dr. Alberton's office.

tion of the external carotid artery loomed on the horizon. He, however, felt that conservative measures could safely be followed for a while and suggested the administration of vitamin K, whole blood, and autogenous blood. The latter was given I. M. for three successive days.

The next day, Jan. 22, the patient voided about 800 cc. of blackish green urine, but urinalysis was negative for the hemoglobin-benzidine test.

Bleeding seemed to be under control for the next few days, but on Jan. 26, the seventh postoperative day, the patient's rectal temperature went up to 104 F. An erythromycin injection was given, and later he bled profusely from both sides of the nose. The next day the rectal temperature went up to 103 F, and 600,000 units of penicillin was given with 0.2 cc. chlorpheniramine (Chlor-Trimeton) maleate because of the patient's allergic background.

On Jan. 28, the packing was removed because of the fever and because the packing was saturated with foul discharge. Bleeding was moderately active, and the nose was packed postnasally and on both sides, with control of the hemorrhage.

On the following day, oozing through the packing was fairly active, and a transfusion of 500 cc. whole blood was given.

Because of the persistence of foul discharge from the nose due to the enforced retention of the packing to control bleeding, a culture was taken. This showed the presence of Gram-positive cocci, streptococci, Gram-negative rods, and cocci. A bacterial antibiotic susceptibility test revealed that the growth of streptococci was inhibited only by neomycin, nitrofurantoin (Furandantin), and chloramphenicol (Chloromycetin). Accordingly, penicillin and erythromycin were discontinued and the administration of chloramphenicol was started.

Bleeding continued intermittently and on Jan. 30, rhinologic consultation was held with Dr. R. C. Martin, who advised to await events and to continue transfusions.

On Jan. 31 the patient had been bleeding bright red blood profusely through the packing and postnasally all during the afternoon. Pulse at this time was 160 and extrasystoles were noted.

Patient was immediately taken to Surgery (6:00 p. m.) and, with the active participation of Dr.

R. C. Martin and Dr. M. J. Groper, the postnasal and left anterior packing was removed without anesthesia. Blood seemed to well up posteriorly, flooding the field, and bleeding points could not be located. An extra large postnasal pack, covered with absorbable gelatin sponge, was applied, with one string drawn respectively through each nasal passage so as to exert firm pressure against the choanae when anchored anteriorly at the columella. The left nasal passage was then tightly packed with several feet of ½ in. plain gauze packing.

While all this was going on, 500 cc. of whole blood was being given to the patient. He was then returned to his room with orders for heavy sedation.

The following day, the patient rested quietly, and intravenous dextrose and 500 cc. of whole blood were administered.

There was no bleeding for the next few days, and the packing was partially removed each day until Feb. 4, 1956, when the packing was completely removed without bleeding.

However, on Feb. 5, the 15th postoperative day, there was a spontaneous, profuse, and brisk hemorrhage from both sides of the nose, starting on the right side. About 18 in. of ½ in. gauze packing in each side controlled the bleeding.

Transfusions were repeated of 500 cc. whole blood. There were episodes of staining of blood on the packing for the next few days. The packing was gradually withdrawn daily until Feb. 11, when it was completely removed without bleeding.

The patient was discharged from the hospital on Feb. 12, 24 days after entry for a simple septum resection. He was seen at the office for the next two weeks and referred to an internist, Dr. E. C. Alberton, for treatment of his secondary anemia.

Hematologic studies at the latter's office indicated the patient's blood picture at this time to be, except for a secondary anemia, entirely normal. Pertinent laboratory data elicited during this period are tabulated in Table 1. Studies of the stained smears were entirely normal, as were also standard prothrombin time tests. The patient was given ferrous sulfate 0.2 gm. q. i. d. under Dr. Alberton's supervision, and in a period of five weeks the red count and hemoglobin had returned to normal limits.

Hospital Readmissions (Service of Dr. E. C. Alberton)

First Readmission: On May 2, 1956, the patient was readmitted to Hahnemann Hospital with the history of bleeding for the past week from an erupting lower right third molar. Despite dental treatment, bleeding persisted. Table 1 gives laboratory data during this and subsequent reentries.

Examination showed redundant granulation, with slow oozing of blood from an erupting right lower third molar. B. P. 120/70, pulse 72; liver edge just palpable on deep inspiration; spleen not palpable.

Because of continued bleeding in spite of local and nonspecific measures, including corticotropin (ACTH) injections, hematologic consultation was requested. The consultant's report follows:

"Twenty-eight year old Puerto Rican male with a history of bleeding from tooth socket. One and one-half years ago, after carrying a bundle, he noticed a large ecchymosis at the right ante-cubital area which came on the next day. Four months ago, at time of submucous resection, had severe bleeding difficult to control. No other history of spontaneous hemorrhage or of easy bruising. No history of drug intake. No definite allergic history.

"Physical Examination: Few cervical and axillary nodes. No hepato-splenomegaly. No jaundice. No telangiectases. Tooth socket looks well at this time. There is a large ecchymosis, right arm at site of needle puncture.

"Impression: Vascular purpura, possibly allergic (eosinophils 6%) although no antigen can be definitely involved at this time from history.

"Recommend: Observation. Anti-histamines or steroids worth a try if necessary for continuous or resumption of severe bleeding."

On May 10, inasmuch as there was no change in laboratory findings and the bleeding appeared to be well controlled, the impacted lower right third molar tooth was removed in Surgery by E. J. Lattig, D. D. S., under lidocaine anesthesia, and the wound was sutured.

Therapy while in the hospital consisted of corticotropin, vitamin C, and antihistamines. Patient was asymptomatic after the tooth extraction and he was discharged May 12.

Second Readmission: On May 20, 1956, the patient was readmitted to the hospital with the history that at 5:00 a. m. his gum started to bleed at the site of the tooth extraction without any evident good reason. The bleeding stopped temporarily after some medicine was given, but recurred and has been steady though slow ever since.

Examination showed bleeding at a slow rate from a granulating surface at the site of the operative wound. B. P. 130/80, pulse 96; liver not enlarged; spleen not palpated.

On May 21 the wound was repacked and resutured by Dr. Lattig in Surgery.

Therapy while the patient was in the hospital consisted of whole blood transfusion, penicillin, corticotropin gel, carbazochrome (Adrenosem) salicylate, diphenhydramine (Benadryl), and vitamin C.

Patient was discharged from the hospital May 24, 1956 at 12:30 p. m., with the diagnosis of bleeding dyscrasia, type undetermined.

Third Readmission: On May 25 at 12:15 a. m. the patient was readmitted to the hospital, about

12 hours after his last discharge, with the history of bleeding recurring (patient stated that it felt like "gushing") at the tooth socket since evening.

Later that day, the wound was cleansed after removal of old sutures by Dr. Lattig in Surgery and, under lidocaine anesthesia, it was repacked and resutured.

Therapy consisted of carbazochrome salicylate, conjugated estrogenic substances (Premarin), penicillin, and fibrinogen. The bleeding stopped after initial intraoral pressure packing, then resumed and became profuse, then decreased to a persistent though intermittent oozing even after the wound had been resutured.

At this time another hematologist was consulted, and on May 31 Dr. P. M. Aggeler saw the patient and made the following notation:

"History of bleeding from gingival flaps on eruption of molar tooth and further bleeding following tooth extraction. Previous history of bleeding following submucous resection. No history of bruising, petechiae, epistaxis, G. I. or G. U. bleeding or of joint hemorrhage.

"Studies done yesterday showed: Clotting time 6⅔ minutes; Quick 1 stage plasma prothrombin 100%; Ware-Stagnell modified prothrombin plasma 100%, serum 20%.

"All the above tests are normal. However, a thromboplastin generation test (Biggs and Douglas) showed grossly abnormal results. When normal plasma was substituted for the patient's plasma in the tests, the results were normal. These results appear to indicate a diagnosis of 'mild' but classical hemophilia. An actual antihemophilic factor titration will be done on the patient's plasma. In severe cases, the level is usually less than 1%. In mild cases, it may be as high as 20%. In view of the above findings, I would recommend treatment with 2-4 units of fresh-frozen (antihemophilic) plasma daily until bleeding has ceased and healing has begun."

The patient was then treated with transfusions of fresh frozen (antihemophilic) plasma on May 28, May 30, June 1, June 2, June 3, and June 4, 1956.

Transient hives appeared after the second transfusion but were controlled by antihistamines. Other therapy consisted of corticotropin, C. V. P. capsules (a bioflavonoid compound with vitamin C), diphenhydramine, and prednisone (Meticorten). Bleeding ceased after the transfusions with the fresh-frozen antihemophilic type of plasma, and the patient was discharged from the hospital on June 6, 1956.

In a subsequent personal conversation with Dr. Aggler I was informed that the antihemophilic factor titration test was 30% of normal. This, he felt, would tend to point to a suggestion of the diagnosis of pseudohemophilia since in true hemophilia the result would be less than 20%.

On Aug. 14, 1956, the patient was hospitalized for six weeks at Mt. Zion Hospital for an infectious hepatitis probably due to the transfusions of fresh frozen plasma. He made an uneventful and complete recovery. There was no recurrence of bleeding during this hospitalization.

Regardless of all studies thus far there has been no positive determination as to whether this patient has a "mild" but classical hemophilia or a pseudohemophilia.

Comment

The problems of management which arose during my conduct of this case were diverse and at times frustrating.

With a negative history and negative laboratory findings and in the presence of a stubborn postoperative nasal hemorrhage, the tendency is first to consider a traumatic cause rather than a blood dyscrasia. So it was more natural to emphasize attempts to control the hemorrhage from the mechanical aspect of surgical trauma rather than to suspect a defect in blood constituents. In the subsequent hospital reentries those working on the case were forewarned and therefore were forearmed.

Then the academic question arose as to, in the event that ligation of the external carotid artery should be indicated, which side should be attempted when the source of hemorrhage appeared to arise from either side? Ligation of the anterior ethmodial artery has been advocated as a simpler means of control, but in this case the source of bleeding could not be pinpointed as just coming from that area.

The hazard of prolonged retention of gauze packing in the nose to control hemorrhage was demonstrated here by the tendency to suppuration, sepsis, and fever. An attempt was made to avoid this retention of purulent secretion by the insertion of F-16 Foley balloon type 30 cc. catheters as postnasal plugs. Such a catheter was inserted through each nostril into the nasopharynx, and the balloons were inflated with air through a 20 cc. syringe to the limit of the patient's subjective tolerance. Each balloon was then pulled up firmly against

and partially through the posterior nares, and both catheters were anchored anteriorly over the columella. However, the bleeding continued, and on removal of the catheters, the next morning, the balloons were found in a collapsed state.

Recourse was had to various and sundry recommended hemostatic agents at one time or another. To name a few: vitamin K; oxalic acid; estrogens; oxidized cellulose (Oxycel gauze) and absorbable gelatin sponge (Gelfoam); autogenous blood and transfusions of whole blood; conjugated estrogenic substances, carbazochrome fibrinogen, and finally fresh-frozen antihemophilic type of plasma. Whether the hemorrhage ceased because of the administration of the last-named agent or because of a spontaneous remission cannot at this time be answered decisively. Sometimes the specific identification of a blood dyscrasia falls within the province of the hematologist. Yet the requirements for differentiation may be so elastic or nebulous as to tax the acumen and resources of even well-qualified hematologists.

When last seen by me in March, 1957, and by Dr. Alberton two months later the patient appeared well and robust with no further episodes of bleeding. As already indicated, there has been no definite determination as to whether this case should be classified as a true hemophilia or a pseudohemophilia. In order to clarify the picture as to the similar and dissimilar features between the two, a discussion of pseudohemophilia (with the previous knowledge of the authors quoted) is herewith appended.

Pseudohemophilia

According to Buchanan and Leavell,[1] pseudohemophilia is a blood dyscrasia in which there is a tendency to bleed associated with a prolonged bleeding time. Clot retraction is usually normal, and the tourniquet test (capillary fragility) is positive in about 50% of cases. The clotting time, the platelet count, and the prothrombin time are within normal limits. This hemorrhagic disorder may occur in either sex, and a familial history may or may not be elicited.

The history or laboratory tests cannot be depended upon to predict the bleeding tendency since the latter does not always correspond with the bleeding time. Convalescence may be uneventful with one major operation while the same patient may hemorrhage profusely at a subsequent operation, or vice versa. Therefore, dental extractions and surgical procedures may become quite hazardous.

In general, a blood dyscrasia may be caused by a deficiency of plasma constituents, a vascular abnormality, or a platelet defect. The conflicting reports from various sources would seem to indicate that, in pseudohemophilia, not all the cases studied have the same hemostatic defect. It is apparent that there may be more than a capillary defect involved in a good percentage of them. At times, a qualitative defect in the platelets is shown by a prolonged bleeding time, impaired prothrombin consumption, a defective clot retraction, poor agglutination of platelets, and morphologically abnormal platelets. Some similarity to thrombocytopenic purpura is suggested by the defective prothrombin consumption test and the variability of clinical severity and bleeding time in the same patient at different times.

There have been conflicting reports about the consistency of results in the prothrombin consumption tests, which have been found abnormal by some authors and normal by others. When the prothrombin consumption test is decreased, one of two assumptions comes to mind. It might be explained by a deficiency of a plasma thromboplastin factor as in hemophilic states or it may indicate a deficiency of the platelet thromboplastin factor as in thrombocytopenic purpura.

It is difficult to classify the patients now diagnosed as pseudohemophilia into a single entity until the influence of heredity and the pathogenesis of this hemorrhagic disorder are more clarified. It is apparent that they

TABLE 2.—*Differential Diagnosis* *

	Psuedohemophilia	True Hemophilia	Thrombocytopenic Purpura	Vascular Purpura
Bleeding time	Prolonged	Normal	Prolonged	Normal
Clotting time	Normal	Prolonged	Normal	Normal
Clot retraction	Usually normal	Normal	Markedly prolonged	Normal
Platelet count	Normal	Normal	Decreased	Normal
Tourniquet test	Positive in 50%	Negative	Usually positive	Positive
Prothrombin time (1 stage)	Normal	Normal	Normal	Normal
Prothrombin consumption	Poor in 50%	Poor	Poor	Normal
Familial	Frequently transmitted by either sex	Sex-linked recessive; transmitted by female to male	May be, but usually is not	No
Sex	Either	Male	Either	Either
Hemarthroses	Uncommon	Characteristic		Occasionally
Petechiae	Rare	Usually absent	Characteristic	Rare
Hematuria	Uncommon	Common		Occasionally
Megakaryocytes in marrow	Normal in number and forming platelets	Normal in number and forming platelets	Increased in number but not forming platelets	Normal in number and forming platelets

* Adapted from Buchanan, J. C., and Leavell, B. S.: Ann. Int. Med. 44:241-256, 1946.

do not resemble other recognized hemorrhagic states. Work still remains to be done to determine whether this disease has a common etiology or whether it is a syndrome with a common hemostatic defect.

Prognosis for life appears relatively good. The incidence in either sex is about equal. In males, the hemorrhagic episodes often decrease in frequency and severity with increasing age, while in females they may become severer after puberty. In either sex, spontaneous remissions may occur regardless of treatment.

The possibility of pseudohemophilia should be borne in mind in the differential diagnosis of every case in which there may be a history of repeated episodes of abnormal bleeding. While the only consistently abnormal laboratory finding is a prolonged bleeding time, the bleeding time on any one determination may be normal. It is incumbent, therefore that repeated determinations of bleeding time should be made in a case in which all other laboratory findings are normal before the possibility of pseudohemophilia is abandoned.

The commonest conditions from which this disease should be differentiated are hemophilia and thrombocytopenic purpura. Vascular purpura is also included in the accompanying differential table in order to show similar and dissimilar features. But these four are by no means the only possible causes involved in the general consideration of the etiology of hemorrhagic disorders. They were grouped together in one category because of the close linkage to the case reported.

There is no satisfactory specific treatment for this condition. The clinical course may be so variable that difficulty may be encountered in distinguishing a good therapeutic result from a spontaneous remission.

Until more is known about the nature of this disease, it would seem that symptomatic treatment and local hemostatic agents would be indicated. All but the most urgent surgery should be avoided. Blood transfusions for anemia would also be in order.

Summary

A case of postoperative nasal hemorrhage is reported.

The problems of hemostasis in the presence of an undiagnosed blood dyscrasia are described.

Subsequent repeated hospital reentries to control an unrelated dental bleeding resulted in the identification of the blood dyscrasia as either a pseudohemophilia or a mild true hemophilia.

Pseudohemophilia is discussed as to its clinical course, differential diagnosis, and treatment.

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Mucous Adenoma of the Nasal Fossa

Report of a Case

R. I. WILLIAMS, M.D., and G. L. SMITH, M.D., Cheyenne, Wyo.

The true mucous adenoma in the nasal fossa, while not a rare tumor, is a relatively uncommon one. References to it in the American literature are scarce and somewhat sketchy; hence, it is felt that the report of this case is of some value to those pursuing the study of this entity along further lines.

The exact classification and behavior of this tumor seems fraught with confusion, apparently because of its being mistaken for other tumefactions containing mucous glands which occur in the nose and paranasal sinuses. Most authors [1,2,4,7] classify it as a benign neoplasm on the basis of its histological behavior but malignant on the basis of its clinical behavior. Natanson [6] classifies the adenoma into two categories, the benign, or true, adenoma, and the malignant, or destructive, adenoma. The first class he describes as being small, encapsulated, and arising most commonly on the tuberculum septi but seen also to arise in other areas where the mucosa is rich in mucous glands, and probably related to inflammatory processes. The second class he describes as being large, rapidly growing tumors, usually filling the entire nasal fossa and seen extending into the paranasal sinuses and cranial cavity in some instances. It is likely that both classes are but two separate stages of the same pathologic entity. Natanson concedes that there are other cases which seem to fall somewhere between the two classes. Ringertz [7] feels that in those cases where malignant change has been observed the malignancy may have existed before and that there is no definite proof of malignant change in an otherwise benign adenoma. While histologically benign and not prone to give rise to regional or distant metastases, this tumor is frequently reported as being locally invasive and prone to recurrence after its removal.[2,7] Its incidence is apparently equal in males and females, and it has been seen occasionally in children but more commonly in middle-aged adults.[7]

The gross appearance of the tumor is characterized by a rose-colored tumefaction, which may be polypous, pedunculated, or sessile and quite friable. Ulceration is uncommon, and when it occurs it is believed to indicate the possibility of malignancy.[1,7] The tumors are most frequently seen in the nasal cavity and rarely in the paranasal sinuses and nasopharynx. The exact site of origin is often difficult to ascertain, but they most commonly arise in the region of the ethmoidal labyrinth. Some have been reported to have arisen on the septum and turbinates.[1,6,7] Extension of the tumor beyond the limits of the nasal fossa is common, and they have been known to erode the lamina cribrosa, the frontal sinus, the sphenoid sinus, and the nasal bones. This apparently occurs, not through invasion or infiltration by the tumor, but by pressure atrophy,[7] and hence the name by which it is often referred, "adenoma destruens." These tumors are often highly vascular, and because of their friable nature they may give rise to brisk hemorrhage upon very slight trauma.

Histologically, the adenoma appears to arise as a hyperplasia of the seromucinous (Bowman's) glands.[1,3] The general histologic picture is a highly variable one from the standpoint of the quantity of stroma,

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proliferation and appearance of the epithelium, and the degree of mucous secretion, but the fundamental structure is fairly constant. Within the group of mucous glands there are frequently seen calcific deposits and bony new growth within the connective-tissue stroma. This metaplastic bone growth may be confused with tumor infiltrating bone, which apparently is never the case with this type of tumor. Cystic glands and papillary ingrowths of epithelium into the glandular lumina are also seen. This epithelium may consist of one or several cell layers. The tumor is usually encapsulated to some degree, but in some instances a well-defined capsule may be a difficult matter to prove. The glandular elements of the tumor appear as a glandular hyperplasia, rich in mucous secretion, and in that respect it may resemble a thyroid adenoma.[2]

In their normal course these are slow-growing tumors whose symptoms are largely mechanical, i. e., those of obstruction, usually of the nasal passages. As the tumor enlarges, swelling about the external nose and sometimes pain, which is seldom severe, develop. Bleeding is a frequent manifestation but is seldom severe unless the tumor is deliberately disturbed, as by biopsy or surgical removal.

The treatment of these neoplasms is surgery, extensive enough to remove completely the entire tumor. This should include the entire base of the tumor even if that means sacrificing the entire lateral wall of the nose. Radiation is of no value.[7] Williams [8] has outlined a very adequate method of surgical exposure of the areas in which these adenomas are prone to arise, which permits complete removal of the tumor mass with a minimum of operative trauma and postoperative disfiguration.

In general, the prognosis is favorable, provided the tumor is completely removed and the diagnosis made early enough, before widespread destruction of neighboring structures has taken place. Again the controversy of possible malignancy arises, and its existence depends upon which author's writings one is reading. Natanson reports one case which he followed for a period of 10 years, during which time a benign adenoma appeared to become truly malignant. He further felt that the lack of reported cases of fatal outcome from this tumor may have been due to insufficient time allowed for follow-up care on the part of the observers. Recurrences following removal of the tumor have been reported frequently in the literature and are thought to be a result of inadequate extirpation of the original lesion,[7] and malignant changes have been reported in these recurrences.[2,6,7] Ringertz [7] feels that such cases may have been malignant from the beginning and that there is no definite proof of malignant change in an otherwise benign adenoma. He also adds that this concept is of little importance inasmuch as the benign adenoma, through expansive growth, can do as much damage as an infiltrating adenocarcinoma and can cause death by local extension as does the adenocarcinoma which has no distant metastases.

We feel that the surgical management of this tumor is the preferred one and that care must be taken in that surgery to be certain of complete removal of the entire tumor. Excision of the tumor by use of a snare, biting forceps, and/or avulsion is not a substitute for excision of the entire attachment of the tumor base. The small amount of deformity of the patient's features and the minor impairment of nasal function resulting from removal of the entire lateral wall of the nasal fossa by a hemilateral rhinotomy approach [8] are a small price to pay to avoid recurrences of this highly destructive and potentially lethal tumor.

The following case from our files is respectfully submitted; it has been presented "in precis" previously.[8]

A 58-year-old white woman was first seen by us on Oct. 8, 1956. At that time she gave a history of obstruction and a sensation of dryness in the right side of her nose for a period of approximately one year. In the six months preceding her initial visit she had been troubled with periodic bleeding from the right nasal fossa. She denied weight loss or headache.

Fig. 1.—Tumor in situ prior to removal. Forceps are pointing to tumor in nose; applicator stick is in right maxillary antrum; lateral wall of nasal pyramid reflected upward and medially.

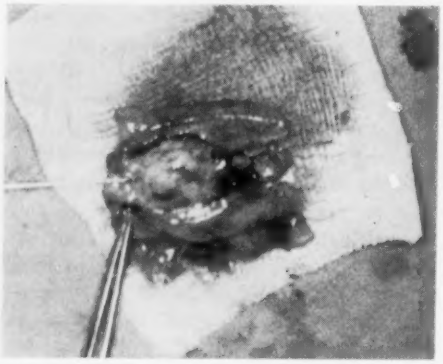

Fig. 2.—Appearance of mucous adenoma, after removal en bloc, with the lateral wall of the nose showing its attachment to the wall of the middle meatus. About half of the tumor had been removed by previous biopsy. Pointer rests on the posterior portion of the tumor.

Upon physical examination positive findings were confined to the right nasal fossa, where a pedunculated, friable, gray-red mass was noted protruding into the right nostril; it bled easily upon contact. Subsequent biopsy revealed the mass to be a mucous adenoma.

On Oct. 26, 1956, a right hemilateral rhinotomy was performed under local anesthesia, and a tumor mass was found arising from the lateral wall of the nose on the right in the region of the ethmoidal labyrinth. The entire right lateral wall of the nose was removed en bloc with the attached tumor mass. (Figs. 1 and 2.) Minimal bleeding attended the surgery, and the postoperative course was smooth and uneventful. The patient was discharged from the hospital on Oct. 30, 1956, and, as of this writing, shows no sign of recurrence.

The histologic examination of this tumor is as follows: "Microsections of the tumor show an incomplete wall of connective tissue lined by respiratory epithelium. Beneath the lining, well-differentiated, orderly mucous glands are present with basal, uniform nuclei and abundant basophilic, mucoid cytoplasm. Focal areas of degeneration are noted in the center of the tumor mass. Focal calcification is also present." (Figs. 3 and 4).

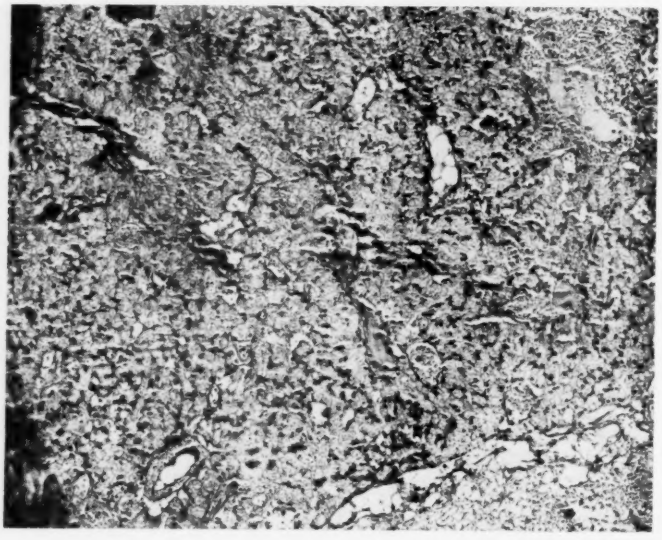

Fig. 3.—Low-power photomicrograph of mucous adenoma. Note focal area of calcification.

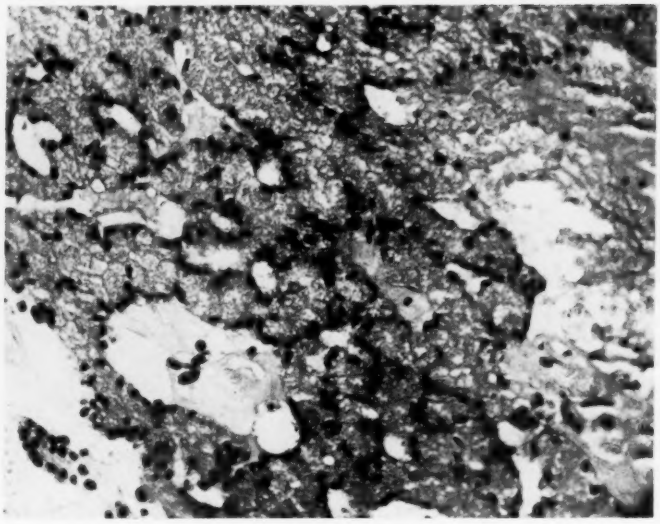

Fig. 4.—High-power photomicrograph of mucous adenoma.

Summary

A summation of the characteristics of the mucous-gland adenoma derived from the literature available to us has been submitted.

Adequate surgical removal has been recommended and stressed for the treatment of this tumor.

A case of a mucous-gland adenoma of the nose has been presented.

S. S. Zuckerman, M.D., Marjory Andresen, M.D., and Henry Y. Tsumagari, M.D., assisted in the preparation of the pathologic material and the photomicrographs for this case.

408 Hynds Building.

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The Importance of the Membranous Nasal Septum

JOSEPH S. STOVIN, M.D., New York

The narrow portion at the lower end of the nasal septum, lying between the cartilaginous septum and the columella, is called the membranous septum, or the septum mobile nasi. As its name implies, it is the most flexible part of the septum. It is formed by a union of the septum mucous membranes immediately below their envelopment of the septal cartilage. This united membrane in turn blends with the skin of the columella. It lies between the semirigid columella and the more rigid septal cartilage. This membrane acts as a buffer between the columella and the cartilaginous septum, softening blows to the lower nose. It may be likened to a shock absorber, absorbing shock-producing blows. It is sufficiently elastic in most cases to prevent the caudal end of the cartilaginous septum from being protruded into the nose as a consequence of severe trauma to the lower nose.

The membranous septum is an important structure concerned with facial expression, especially that elicited by speech and laughter. A normal membrane allows the nasal muscles to function properly. In repose an absent or rigid membranous septum is not of great importance. But since it is involved in varying degrees in all facial expressions we see how important the mobility of this portion of the septum is to the patient's appearance as well as his physical comfort. Removal, partial or complete, of the membranous septum results in rigidity which causes an unpleasing appearance and usually stamps the patient as a "plastic nose" regardless of how pleasing the appearance is in repose. Thus we see how imperative it is for us to treat this part of the septum with great care during operations on the nasal septum and on plastic procedures on the external nose.

In the conventional submucous resection of the septum the membranous septum is not of much importance, especially where there is no obstruction at the caudal end of the cartilaginous septum. This operation usually commences at least ¼ in. beyond the end of the cartilage and in no way involves the membranous portion. Where there is dislocation of the cartilage into a vestibule or an anterior septal obstruction, I prefer to do the operation which I call septoplasty.[1] Only one incision is made along the length of the protruding cartilage, and through it the mucous membranes on each side of the septum are elevated. When proper care is exercised no damage is done to the membranous septum. Through this procedure a generous removal of the septal cartilage can be done with no fear of a subsequent saddling of the external nose, and all obstruction due to cartilage can be removed. The bony septum is treated by removal or fracture, depending on the findings. The removed cartilage is trimmed and sutured in place between the flaps, and the original incision at the junction of the cartilaginous and membranous septum is sutured together. This prevents the contracture which results in saddling and also gives the patient a more rigid septum, one which will not sway with respirations, one of the objections to the conventional submucous resection.

However, this desired rigidity of the septum should not include the membranous portion. I feel that the procedure of transfixion of the septum, except when done in conjunction with a rhinoplasty, contributes to trauma of the membrane, causing scarring and loss of flexibility. The resulting contracture may cause slight shortening of

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the nose. This would give it a pudgy appearance, which can only be avoided by elevating the nasal skin and possibly treating the lateral cartilages, a procedure followed in rhinoplasty. This is necessary to allow proper draping of the skin, which in turn gives the nose a pleasing appearance.

In rhinoplastic procedures requiring a transfixion of the septum no damage to the membranous septum will occur if we exercise care. By making traction on the membrane either manually or by traction sutures one can make the incision along the very edge of the cartilage, and none of the flexible septum will be sacrificed. It can be sutured to the cut edge of the cartilaginous septum with no loss of its flexibility.

In cases where a columellar strut or batten is necessary the graft can be inserted from above down into the pocket, starting at the juncture of the two medial crura of the lower lateral cartilages. In this way another incision in the side of the columella is avoided, with less chance of contracture or narrowing of the membranous septum.

Summary and Conclusion

The membranous septum is anatomically very small in comparison with the other parts of the nasal septum. It is, however, of great importance during surgical procedures, either septal or rhinoplastic. Removal of or trauma to it may result in great discomfort to the patient. In addition there may be a displeasing appearance, especially during changes in facial expression. It behooves the surgeon to use diligence and care to preserve this important structure.

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Prevention of Hemorrhages in E. N. T. Surgery by Interstitial Application of Thromboplastin

MAHMOUD KAMAL MUFTIE, M.D., Basrah, Iraq

The preoperative treatment for preventive hemostatis consists of supplying to the patient all necessary factors playing a role in blood clotting, and capillary retraction. Calcium salts, vitamin K preparations, different preparations of thromboplastin, blood transfusion, etc., are most commonly used. Vascular retraction and elasticity factors, such as vitamin C and P are also indicated in patients suffering from vascular rigidity and fragility.

In minor and less extensive operations in otorhinolaryngology, if local anesthesia used, the epinephrine and epinephrine-like substances produce the necessary ischemia of the operative field. Even if the vasoconstrictor activity stops during operation, a local application of epinephrine is indicated. The disturbing element in this case is the reflex hyperemia which starts after epinephrine action is arrested. Local application of different preparations containing thromboplastin help very much in obtaining a dry field, and prevent blood loss in cases of diffuse bleeding. Unfortunately there are few indications as to where a thin surface layer of precipitated fibrin can resist blood pressure and prevent a secondary hemorrhage. The use of diathermy helps a great deal, but the indications are very limited as it causes deep necrosis.

I used Clauden (Luitpold Werke, thromboplastin) mixed with procaine for infiltration anesthesia in tonsillectomy. It seems that ischemic conditions found with this method are due to the intravascular clotting of blood, and therefore I proceeded to investigate the method from the physiological point of view. Wooldridge [2] discovered that intravenous injection of thromboplastin easily causes intravascular clotting. Eagle [3] obtained intravascular clotting by intravenous injection of trypsin, certain proteolytic snake venoms, and by intravenous injection of 1 mg. of thrombin. Howell's theory assumes that the thromboplastin neutralizes heparin, which normally maintains the fluidity of blood in vivo, so that calcium ions convert prothrombin to thrombin. Thrombin is an unsaturated compound which is capable of combining with more fibrinogen to form fibrin. He compares these actions to the actions of digestive enzymes on proteins. Fibrinogen is first precipitated as fibrin by union of thromboplastin and thrombogen (prothrombin). This fibrin is then hydrolyzed and dissolved by further action of enzymes.

In every case the initiation of the act of clotting depends on the addition of thromboplastin to the plasma. The formation of the thrombi can be prevented by adequate slow injection of heparin. The modern tendency, however, has been to attribute the fluidity of blood in the vessels to the action of an anticoagulant factor such as the presence of heparin in blood. In this study I would demonstrate the laboratory and clinical methods and results concerning intracapillary blood clotting and its use as preventive hemostasis in otorhinolaryngological surgery.

Experimental Studies

Determination of the thromboplastin-heparin titer and their interaction was the first objective of the experiment. It consisted of two series of tests: test tube and nylon capillary tube blood-clotting tests. We

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used heparin (Nordman Werke G. M. B. H. 1000 I. U. per milliliter) and various commercial preparations of thromboplastin merely in order to ascertain the most powerful one (highest titer of thromboplastin). These preparations were the following: Coagulen (Ciba); Clauden (Luitpold Werke); Thrombin, Topical (Parke, Davis); Thrombase (Roussel); Hemostatique Erce (Carriere); Manethol (Bayer).

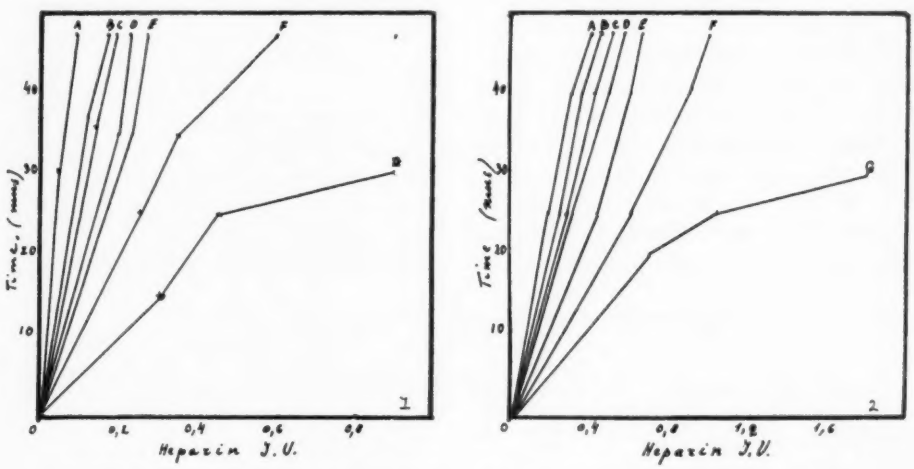
In the first series of experiments the dilutions of heparin were prepared in concentrations from 0.00003 to 500 I. U. per milliliter. To each milliliter of dilutions 0.1 ml. of each of the above mentioned thromboplastin preparations was added; then each test tube received 1 ml. of freshly drawn blood, and the test tubes were left in an incubator at 37 C and checked for clotting reaction every minute. For the second series we made the same arrangement of dilutions but in paraffined test tubes, from which immediately after mixing of components the contents were aspirated in capillary (0.1 mm.) nylon tubes (polyethylene catheters), each 12 cm. in length. Every five minutes a 4 mm. piece was cut by a paraffined razor blade and examined for clotting with a glass capillary tube. The results of the two series of experiments are shown in graphs 1 and 2.

It has been shown that there is a great difference among various commercial preparations of thromboplastin in neutralizing heparin effect. The most powerful preparation was Hemostatique Erce. Only 0.1 ml. of this preparation in one-half hour inhibits about 2 I. U. of heparin. For that reason we used this preparation in our clinical investigations.

Clinical Investigations

It is well known that hemostasis by means of epinephrine is of very short duration, and for the most part not satisfying. In spite of several applications of epinephrine in the operating field, bleeding does not stop as expected. Increased fluidity of blood, caused by overconcentration of heparin, decreases the normal concentration of thromboplastin in corresponding tissues, causing prolonged hemorrhages. Postoperative bleeding, surgically uncontrollable, and especially the crucial problem of secondary bleeding, are the main problems in otorhinolaryngological surgery.

I chose three of the commonest operations in E. N. T. practice for the experiment concerning biological preoperative hemostasis by intracapillary blood clotting. The method was quite simple. To the local anesthetic



Graphs 1 and 2.—*A*, control. *B*, Thrombase. *C*, Manetol. *D*, Thrombin, Topical. *E*, Coagulen. *F*, Clauden. *G*, Hemostatique—Erce.

(procaine HCl) we added a thromboplastin preparation (Hemostatique Erce) with or without epinephrine, with or without hyaluronidase. The mixture was injected, infiltrating the tissues in tonsillectomy, submucous septum resection, turbinectomy (inferior or middle). Those operations are very hemorrhagic and every one has its irreducible minimum of blood loss.

The following four mixtures were used in our experiments:

A. 1% procaine HCl plus Hemostatique (aa)
B. 1% procaine HCl plus epinephrine plus Hemostatique (aa)
C. 1% procaine HCl plus hyaluronidase (1000 I. U. B.) plus Hemostatique (aa)
D. 1% procaine HCl plus hyaluronidase plus epinephrine plus Hemostatique (aa)

The quantity of Hemostatique was always equal to the total volume of the other parts of the mixture. In tonsillectomy the mixture was injected in the usual classic points: anterior pillar, posterior pillar, and retrotonsillar area. In submucous septum resection the mixture was injected between the perichondrium and mucous membrane, and in turbinectomies in corresponding leading points according to the case. In every case we verified by aspiration that the needle was not intravascular.

The control cases were treated by the usual procaine HCl-epinephrine infiltration. During operations use of tampons was avoided; blood was aspirated by aspirator and conserved in graduated vessels. Also in case of bleeding after operation aspiration was continued with suction, and the blood was again collected. The quantity of collected blood was the criterion for the application of hemostatic measures. The statistical probability for each method, and for each kind of operation, was calculated by Thün's formula: $\sqrt{a \cdot p}$, where "p" was the median value of blood lost in control cases and "a" the median value of the blood lost by the method and in the kind of operation under investigation. The Table shows the results obtained in our department during eight months. By use of the mixture "D" at quite bloodless operations and a dry field were attained. The other mixtures were also effective in reducing bleeding volume. Sometimes there was not more than 0.5 cc. of blood during one tonsillectomy by dissection. Turbinectomies, well known as hemorrhagenic operations, were converted to dry operations without any secondary bleeding at all, even during removal of the first pack.

The healing of wounds was not disturbed by the applied mixture, and there was no delay observed in comparison with control cases. Up till now we have not been able to observe any complications, local or general, which can be the result of thromboplastin infiltration.

Comment

Every tissue contains some quantity of thromboplastin, in different concentration. In an intact tissue thromboplastin is in inactive form, and if the tissues are injured it takes some time for liberation of thromboplastin. In cases of thromboplastin deficiency in the tissues the bleeding during and after operation can be very abundant. In fortifying the thromboplastin content of the tissue in the operative area by infiltration with a thromboplastin preparation, we decrease the possibility of bleeding during and after operation. The intracapillary blood clotting was observed in histological prepara-

Results Obtained with Interstitial Application of Thromboplastin in Various Operations

Operation	I		II		III		IV		Controls
	Mv	Tv	Mv	Tv	Mv	Tv	Mv	Tv	Mv
Tonsillectomy	15	42.9	11	36.7	5	27.8	3	19.2	123
Turbinectomy	12	27.9	10.5	80.2	8	22.07	2.9	42.09	61
Septum resection	8	19.1	5	15	2.5	33.4	0.5	4.99	45

Mv = median value of collected blood in cubic centimeters.
Tv = Thün's value: $\sqrt{a \cdot p}$; a i's control Mv, and p is investigative Mv. This value represents statistical probability of significance.

tions of dissected tonsils and resected turbinates. The safety of the application of thromboplastin interstitially (but not intravascularly), was observed in more than 78 operations. Pálos [4] and Kocsan found that thrombin can be oxidized and in its oxidized form become inactive. It means that the catalytic action of thrombin is dehydrogenatic, and that oxidation inhibits its action. According to the same authors, heparin acts as catalyst in oxidation of thrombin. Lison [5-7] considered that a specific reaction of sulfuric esters of high molecular weight, as in the case of heparin, is due to the adsorptive power of negatively charged particles of mucoitin acid. Calcium interferes there probably as inhibitor of heparin-like esters by precipitating sulfuric acid anions. The processes of liberation of thromboplastin from tissues, the maintenance of thromboplastin-heparin balance, and the inactivation of thromboplastin still remain unknown. It is self-evident that an interstitial infiltration by thromboplastin inhibits excess of heparin-like substances locally present and gives excellent opportunity for control of operative and postoperative bleeding in many surgical interventions, as we observed in various otorhinolaryngological operations. Finally, I must repeat that no harmful effect was observed, and no accident was recorded. Great care must be taken to avoid intravascular introduction of thromboplastin, which can be extremely troublesome in case of an intravenous injection.

The problem of bleeding during operation and the difficulties in controlling postoperative bleeding during the first days after intervention are well known to every laryngologist. For the first time this kind of surgery resembles abdominal surgery in which bleeding is completely controlled by the surgeon. This method enabled me to shorten the duration of the operation and to follow under good visibility in a dry field all instrumental handling. I hope in the future to perfect the application of the method and thus make the results more certain.

Summary and Conclusions

By interstitial application of thromboplastin preparations I succeeded in controlling bleeding and obtaining a dry field in several otorhinolaryngological operations, known as very hemorrhagenic ones. The comparative estimation of thromboplastin value in various commercial preparations was made by laboratory methods, and the highest titer was obtained in Hemostatique Erce. Mixed with a local anesthetic, thromboplastin was injected in to the operative area, and the operation was performed. A comparison of the amount of aspirated blood (by suction) in the operation done with thromboplastin infiltrations and in control cases shows the great advantage of the method in control of bleeding during operation as well as secondary bleeding. The method was completely harmless.

Royal Hospital.

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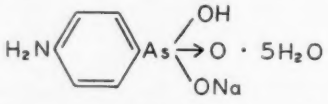
The Toxic Action of Some Compounds on the Inner Ear and Its Central Vestibular Connections

An Experimental Investigation with Reference to Nitrogen Mustards and Sodium Arsanilate (Atoxyl)

HERMAN DIAMANT, M.D., Stockholm

Both the peripheral and the central part of the vestibular apparatus can be specifically affected by a number of substances, chief among which are perhaps arsenical compounds, streptomycin, and nitrogen mustards. As early as 1907 Ehrlich, who at the time was investigating the action of various arsenical chemotherapeutic agents upon trypanosomes, observed a peculiar symptom in mice following injection of arsacetin, an arsenic derivative. In his experiments he used 1 cc. of a 1:166 solution of arsacetin, and found incidentally that some of the mice injected in this way developed waltzing symptoms highly reminiscent of Japanese waltzing mice. On elevation of the dose to 1 cc. of a 1:30 solution, the symptoms occurred regularly and could have a duration of eight to nine months. Ehrlich did not study any lesions that arsacetin might have produced. Röthig (1909), on the contrary, made a detailed histologic examination of the central portion of the vestibular apparatus, and observed distinct degeneration of the vestibular nerve in both old and young mice. He did not state, however, whether he had found lesions of the peripheral organ. Further investigations have since been conducted with various arsenical preparations. Scheinker (1944) studied lesions of the central nervous system in five cases of encephalopathy due to arsphenamine, and found chiefly vascular changes of the type associated with central vasoparalysis. He suggested that the phenomenon might be specific for arsphenamine.

Davini (1950) showed that dichlorophenolarsenic acid, too, induced vestibular symptoms and reported having found pronounced lesions in the supravestibular pathways as well as in the extrapyramidal system. Nassuphis (1951) studied the action on the inner ear of sodium arsanilate (Atoxyl), which has the structural formula:



Sodium arsanilate was administered either parenterally via a subcutaneous injection, or locally in the ear via injection through the tympanic membrane. The animals were killed and examined histologically as soon as the pinna reflex had subsided. After both modes of administration there were distinct lesions in various parts of the inner ear. Nassuphis did not examine the brains.

Arsenical compounds were, accordingly, the first substances that were shown to have a specific vestibular action, at all events insofar as the vestibular organ was damaged by a substantially smaller dose than was the rest of the body. A number of substances with a specific affinity for the vestibular apparatus have since been found.

One important group of these ototoxic substances are the nitrogen mustards. Goldin, Noe, Landing, Shapiro, and Goldberg (1948) demonstrated a waltzing syndrome in mice after injection of heterocyclic or dialkyl-β-chloroethylamines. Similar experiments had

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From the Ear, Nose and Throat Department (Head: Prof. T. Skoog, M.D.), Karolinska Sjukhuset.

been conducted by Krop, Wescoe, Goldin, and Landing (1947), who found marked neurologic symptoms with distinct vestibular features. The histologic changes were localized to various groups of ganglion cells, and in particular to the Purkinje cells. In the late stage there was demyelination in the cerebrum, central ganglia, and brain stem. No lesions were observed, however, in the inner ear.

Diamant (1954) showed that nitrogen mustard (tris [2-chloro-ethyl] amine hydrochloride, mechlorethamine hydrochloride), in a dose of 3 mg. per kilogram of body weight, had a destructive effect on the labyrinth and abolished the labyrinthine responses. This was manifested in the absence of a vestibular syndrome after intracarotid injection of cholinesterase inhibitors. Histologic examinations of the brain revealed normal conditions. It was also found that nitrogen mustard produced no change in the cholinesterase activity or in the blood-brain barrier.

In addition to the above-mentioned substances which have a more or less specific action on the vestibular organ, there are several others which affect that organ in varying degree as well as the rest of the body. Foremost among them, not least from the clinical point of view, is streptomycin. Other ototoxic agents are certain thiuram sulfides, of which disulfiram (tetraethylthiuram disulfide [Antabuse]) may produce disturbances of balance (Hanzlik and Irvine, 1921). Brug and Funcke (1951) showed that disulfiram administered orally in guinea pigs, completely abolished the vestibular reaction. Miehlke (1952) found that ammonia had induced a vestibular disturbance in one case; and in an experimental investigation prompted by this finding, he observed that the primary lesion was probably localized to the vestibular nuclei in the brain stem. Napolitano and Longo (1952) reported a number of substances, including mephenesin (Myanesin) that had a more or less inhibitory effect on the vestibular function, though without abolishing it completely.

The literature dealing with the toxic action of streptomycin on the acoustic nerve is extensive; yet it still remains to be established whether streptomycin has a central or a peripheral effect. Floberg, Hamberger and Hydén (1949) claimed to have demonstrated lesions of the vestibular nuclei by a microspectrographic method. Their results were endorsed by Jongkees and Hulk (1950), whose cupulometric studies pointed to central damage. Berg (1951), on the other hand, found that the lesions produced by streptomycin were localized chiefly to the peripheral organ. Skoog (1950) conducted investigations in which he elicited Forssman's intracarotid syndrome and found a reaction in the animals although the function had been abolished by streptomycin. He concluded that it was probably a case of peripheral damage, at all events in the final stage of the destructive effect of streptomycin. A similar opinion is held by other authors, including Femenic and Salaj (1953.)

In the last few years a number of investigations into the vestibular mechanism have been conducted at the Ear Department of Karolinska Sjukhuset (Floberg, Hamberger and Hydén, 1949; Diamant, 1952; Floberg, 1953; Floberg and Koch, 1954; Bergström and Koch, 1956). In attempts to influence syndromes elicited from the vestibular apparatus by injection of nerve gases producing chemical destruction of the vestibular nuclei, various ototoxic agents were used (Diamant, 1954). In those experiments it was chiefly nitrogen mustards and sodium arsanilate that had an appreciable destructive effect. The results obtained then have now been implemented by further experiments with the substances in question. The aim has been to correlate the action of these greatly differing compounds; at the same time a correlation of this kind might contribute to our understanding of the mechanism of the vestibular organ.

Material and Methods

The experimental animals were guinea pigs, the weight of which varied but was

usually about 400-500 gm. The experimental technique has already been described in principle (Diamant, 1954). An intracarotid injection was given on the right side and directed toward the heart, with simultaneous occlusion of the left carotid. Owing to the guinea pig's anatomy, the injected substance reached the vestibular region on the right side. The guinea pigs were examined, before use, with respect to hearing and vestibular reaction. The hearing was tested by striking a tuning fork and observing the animal's response. If the guinea pig showed a pinna reflex to a rather faint note, it was considered to have satisfactory hearing. The animal was then placed on a rotating plate and subjected to 10 complete revolutions over a period of 20 seconds in each direction, after which it was considered sufficient to observe the presence of postrotary reactions falling within the normal range. In some cases the tipping reaction was employed for establishing whether the vestibular function had been totally abolished. More detailed tests than these were felt to be unnecessary. After injection of the ototoxic agents the hearing and the vestibular organ were tested repeatedly at varying intervals. In this way it was possible to find out if and when the hearing disappeared.

Agents Employed.—The following substances were used:

1. Nitrogen mustards
   - A. Dimethyl-2-chloroethylamine hydrochloride (N_1)
   - B. Methyl-bis(2-chloroethyl)amine hydrochloride (N_2)
   - C. Tris(2-chloroethyl)amine hydrochloride (mechlorethamine hydrochloride) (N_3)
2. Sodium arsanilate

The dosages of these substances varied substantially, as shown in the tabulated results.

In addition to the nitrogen mustards and sodium arsanilate used in these experiments, injections of nerve gases, Tabun (ethyl dimethylphosphoramidocyanidate) and Sarin (isopropyl methylphosphonofluoridate) were also given. These cholinesterase inhibitors produce vestibular syndromes of central origin (Diamant, 1954). An elicited syndrome may be taken as proof of a functioning vestibular mechanism.

TABLE 1.—*Number of Guinea Pigs Injected with Nitrogen Mustards*

Dose in Mg/Kg Body Weight	Number of Cases		
	N_1	N_2	N_3
1		1	10
2		8	22
3			25
4			3
5	2	6	4
8-10	2	8	4
18-25		2	
50-60			2
120-150	2		1
Total	6	25	71

Results

Nitrogen Mustards.—A total of 102 experiments were conducted, in all of which the nitrogen mustard, dissolved in salt solution, was administered by intracarotid injection with a technique that has been described earlier. The toxicity of nitrogen mustards rises with the number of chlorine atoms, and N_3 has a far greater toxic effect than the other two. The dosages used here are shown in Table 1.

Only six experiments were performed with N_1, which was found to have no effect on the organ of hearing or balance even in a dose as high as 150 mg/kg.; at all events, no effect was demonstrable during the brief survival time after this large dose.

An analysis of the results obtained with N_2 is presented in Table 2. Doses of 1 and 2 mg/kg., it will be seen, did not appreciably influence the balance or hearing. Nor was any such influence observed in the six guinea pigs injected with 5 mg/kg., though in one animal which received that dose after

TABLE 2.—*Results of Injection of N_2*

Dose in Mg/Kg Body Weight	Number of Cases	Cases with Labyrinthine Deafness	Effect on Intracarotid Syndrome *
1	1	0	None
2	8	0	None
5	6	0	Abolished in 1 case
8	1	0	Not tested
10	7	4	Abolished in 1 case
18-25	2	2	Not tested

* After injection of 0.03 mg. Tabun/kg. body weight (Diamant, 1954).

TABLE 3.—*Results of Injection of N_2 Followed by Intracarotid Injection of an Irreversible Cholinesterase Inhibitor*

Dose N_2 Mg/Kg	Number of Cases (All with Labyrinthine Deafness)	Positive Syndrome After Injection of Inhibitor *
1	5	5
2	12	12
3	18	4
4	3	10

* 0.03 mg. Tabun or 0.02 mg. Sarin per kilogram body weight.

an intracarotid syndrome had been elicited with Tabun, the syndrome was rapidly abolished. In four of seven that received 10 mg. of N_2 per kilogram, labyrinthine deafness ensued. Three guinea pigs were injected with Tabun, which produced a normal syndrome even though two of the animals had labyrinthine deafness. In one animal 10 mg. of N_2 per kilogram abolished a vestibular syndrome elicited with Tabun.

Injection of N_3 produced labyrinthine deafness in all instances. When an inhibitor was administered after injection of 1 and 2 mg/kg., it did not prevent an intracarotid syndrome. If the preparatory dose was raised to 3 mg/kg., a syndrome developed in only 4 of 18 cases. With a further rise of the dose to 4 mg/kg. the syndrome was not elicitable. Only three experiments were performed with this dosage. In the animals that received more than 4 mg/kg. no further experiments were possible because of death very soon after the injection. The intervals between the onset of labyrinthine deafness and death were very short.

In three of the four guinea pigs that first received 1 mg. N_3/kg., then an injection of Tabun, and developed a typical syndrome, a further 1 mg. N_3/kg. was injected. It rapidly abolished the syndrome. Similar experiments were performed on six animals that had received 2 mg/kg., and here, too, the syndrome was rather speedily abolished.

In all experiments with nitrogen mustard the interval between injection of the latter and of the cholinesterase inhibitor was dependent on the time taken for labyrinthine deafness to develop. With the higher doses of 3 and 4 mg/kg. this was delayed a few hours, but with the lower doses it could

TABLE 4.—*Results of Injection of Sodium Arsanilate*

	Dose in Mg/Kg Body Weight				
	25	50	60–80	100	150–200
Reaction					
0	2	2 (2)	1	1	
(+)	1	4	8 (2)	3	1 (1)
+		1	1 (1)	6 (2)	7 (2)
Total	3	7 (2)	10 (3)	10 (2)	8 (3)

The figures in parentheses denote the number of cases with syndromes after injection of an inhibitor.
0=No effect on the labyrinth.
(+)=loss of pinna reflex but normal vestibular reaction.
+=total labyrinthine destruction.

take up to two days. In each instance the hearing was affected first. The pinna reflex sometimes disappeared within a few hours, even at a dosage of 1 mg. N_3/kg. The labyrinthine deafness was checked with tilting and rotation. The caloric reaction, on the other hand, usually was not attempted; it was found difficult to evaluate. This test is hazardous, moreover, in that it often gives rise to otitis associated with labyrinthitis.

Sodium Arsanilate.—Sodium arsanilate, as will be seen from Table 4, was injected in a total of 38 animals in dosages ranging from 25 to 200 mg. per kilogram of body weight. Total labyrinthine deafness was elicited only in occasional instances with doses of 25 and 50 mg/kg., and still failed to occur in some cases after doses of between 60 and 80 mg/kg., although the hearing was abolished very early. At doses of 100 mg/kg. the labyrinthine reaction was totally extinguished in 6 of 10 cases. Two of these six animals received an intracarotid injection of a cholinesterase inhibitor (Tabun), which produced a typical syndrome. At even higher doses the labyrinthine function was very swiftly abolished in seven of eight animals. Unfortunately these doses were highly toxic, the guinea pigs dying after only 12 hours. It was essential, therefore, to give the intracarotid injection the same day, and this was done in two of the seven animals where the labyrinthine function had been abolished. In both instances an unequivocal syndrome developed. Further experiments with sodium arsanilate have served to verify these figures. The experiments in question are part of a series designed to elucidate the central action

of cholinesterase inhibitors, and will not be reported here. In no experiment has labyrinthine deafness been produced where injection of an inhibitor has failed to elicit an intracarotid syndrome.

Comment

The two groups, nitrogen mustards and sodium arsanilate, present major differences, chiefly with regard to chemical structure and mechanism of action; but they have ototoxicity in common. Nitrogen mustard is notably a highly potent cell poison and, as such, has been used chiefly in some neoplastic diseases. Some of the substances, administered in adequate doses, produce waltzing symptoms in mice. Goldin et al. (1948) found circumscribed though distinct lesions of the brain stem in mice injected with N_2. They did not try N_3, but nitrogen mustards with a similar formula produced neither waltzing symptoms nor histologic changes in the cerebellum or brain stem. It is difficult, however, to decide whether these substances are comparable with N_3. In no animal with brain lesions did Goldin et al. observe any histologic changes in the inner ear, notwithstanding the presence of marked symptoms. In their opinion, the reason that some of the substances with a higher chlorine content failed to elicit waltzing symptoms lay in their high toxicity, which caused death before symptoms of cerebral damage had time to appear. Goldin et al. also studied arsacetin and sodium arsanilate. They verified Ehrlich's observation of waltzing symptoms in mice after injection of the former compound, but were unable as a rule to elicit symptoms with sodium arsanilate, even though they used high doses. After injection of sodium arsanilate and arsacetin there was no demonstrable cerebral damage resembling that after nitrogen mustard.

Sodium arsanilate, which, like nitrogen mustard, is a cell poison but acts chiefly upon highly specialized cells, early came into use as a chemotherapeutic agent against diseases due to trypanosomes. It has a specific action on the optic nerve and the sensory cells of the eye (Fischl and Schlossberger, 1934). As Nassuphis (1951) has shown, it also exerts a specific effect on the inner ear, with major lesions even after such relatively small doses as a total of 250 mg/kg. injected over eight days. The lethal dose (single dose) is 75 mg/kg. for guinea pigs. Goldin et al., it may be pointed out, injected doses totaling 960 mg/kg.—approximately double the lethal dose. Incidentally, it was already reported in E. Merck's Jahresbericht of 1911 that repeated small doses of sodium arsanilate lead to blindness and deafness in dogs. There are no reports of investigations demonstrating conclusively that sodium arsanilate produces central damage, but other arsenic compounds such as dichlorophenoarsenic acid have been stated to produce supravestibular lesions in mice that have shown waltzing symptoms after injection (Davini, 1950). In this connection Peters (1955) emphasizes the importance of bearing in mind the greatly divergent biochemical and clinical effects of different arsenic compounds.

We are thus concerned with sodium arsanilate, which is highly ototoxic but acts solely upon the peripheral vestibular organ, where it produces microscopic lesions. Nitrogen mustard causes not peripheral but central lesions, although they are not always histologically demonstrable with conventional methods. The above experiments demonstrate that this substance has a peripheral effect, too, and produces labyrinthine deafness. Neither of these two agents has any effect on the cholinesterase activity. A comparison with two other substances known to have an ototoxic action may be worth while for studying the possibility of a common denominator of the effect.

Streptomycin—Streptomycin probably has both a peripheral and a central action. Not even in large doses or when applied locally does it produce any acute symptoms. Nor, as far as is known, does it reduce the cholinesterase activity. Investigations have been reported which suggest that while its periph-

eral effect is irreversible, its central action is not (Skoog, 1950).

Nerve Gases.—Nerve gases produce acute symptoms but have no peripheral effect. They act strongly upon the cholinesterases but cause no histologically verifiable lesions.

For comparative purposes, Forssman's antigen might be considered too. Its sole effect lies in breaking down the blood-brain barrier, without affecting either the labyrinthine organ or the cholinesterase activity. It gives rise to acute symptoms but does not cause histologic changes.

The nitrogen mustards and sodium arsanilate and streptomycin are ototoxic substances that produce histologic lesions. The chief localization of the last, however, has been much discussed. Localization is doubtless central for nitrogen mustards but, in all probability, peripheral for sodium arsanilate. Streptomycin, it would seem, may give rise both to central and to peripheral lesions in the labyrinthine organ; yet the former lesions appear to be reversible. None of the substances mentioned here have any effect on the cholinesterases, which are inhibited on injection of nerve gases. The typical vestibular syndrome resulting from injection of Tabun can also be induced by a corresponding intracarotid injection of Forssman's antigen, as well as by injection of N_3 in large doses. However, it is atypical and of fairly short duration, seldom lasting more than five minutes.

It seems impossible at the present time to find the mechanism of the ototoxic effect, whether it consists chiefly in destruction of different parts of the labyrinthine organ or irritation of its central portion, resulting in a vestibular syndrome.

Summary and Conclusion

In the experiments reported here, intracarotid centripetal injection of sodium arsanilate (Atoxyl) and nitrogen mustards demonstrated once more the ototoxic effect of those compounds. Nerve gases were similarly injected in the hope of establishing whether the toxic effect was central or peripheral, for with this mode of administration they are known to elicit a central vestibular syndrome. Sodium arsanilate, it was found, probably had a largely peripheral effect, but nitrogen mustard caused, in addition, destruction of the vestibular nuclei. No common denominator can yet be found for the action of the different ototoxic agents.

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Congenital Malformation of Incus

H. D. BROWN KELLY, M.D., Glasgow

Malformation of one ossicle in an ear which otherwise appears to have developed normally is extremely rare. Most of the reported cases with deformed ossicles are those of congenital atresia of the ear, where the ossicular abnormality is accompanied by atresia of the canal and other deformities of the middle and inner ear. In this type of case fusion of malleus and incus is frequently observed. Shambaugh (1952) records that in 22 surgically treated ears with anomalies of the sound-conduction apparatus, 15 had atresia of the external canal. In 10 the malleus and incus were malformed, and in 5 there was an abnormality of the stapes.

Altmann (1951), in a comprehensive review of malformations of the hearing organ (with 250 references), stresses the great rarity of isolated ossicular deformities and maintains that the stapes is the ossicle most frequently affected. He also draws attention to the rarity of malformations of the articulations between the auditory ossicles as solitary phenomena.

Report of a Case

A woman aged 29 was referred by Dr. Forbes Walker of Ayr, regarding deafness. This was first noticed at the age of 18 years, and had progressed slowly, the right ear being the worse affected. Bilateral tinnitus had been present for several years, most marked at the time of her menstrual periods. At these times she also experienced slight vertigo. No paracusis Willisii was noted. She had borne two children and had not noticed any particular increase of deafness during or immediately following the pregnancies. There was no history of earache or aural discharge. Tonsils and adenoids had been removed at the age of 6 years. Her mother suffered from bilateral deafness which had been diagnosed as otosclerosis.

On examination, nose, nasopharynx, and pharynx appeared normal. The drumheads were intact, slightly indrawn, and of good quality. Tuning fork tests and audiograms suggested otosclerosis with a loss of about 40 db. for air conduction in each ear. Bone conduction was good, there was a good "step" between bone and air conduction, and no drop in hearing for the high frequencies.

Dr. Samuel Rosen, who was visiting Glasgow at the time, operated on her left ear in the Victoria Infirmary, on Jan. 26, 1956. He found a break in the ossicular chain in the form of a small gap between the long process of the incus and the head of the stapes. The long process did not appear unduly short, but was short enough to avoid contact with the stapes. An attempt to bridge the gap was made by inserting a small piece of meatal skin and subcutaneous tissue so that the ossicular chain was restored. The stapes was fixed, but mobilized fairly easily at the neck.

One year later, the hearing showed an improvement of 15-20 db. in the left ear, and she requested that the right ear be operated upon.

This was carried out on Dec. 4, 1956. On opening the middle ear through the Rosen approach, I was surprised to find no sign of a long process of the incus. The stapes, apparently normal in appearance, stood isolated. It moved on applying a sharp probe to its head. An attempt was made to plaster the membrane directly onto the stapes, as it was hoped thereby to obtain a columella effect. Unfortunately no improvement in hearing resulted; in fact, some postoperative drop in the middle register was noted.

Embryology

In order to understand the varieties of ossicular malformations, some embryological points should be considered. The external auditory meatus and the auricle develop from the first branchial groove. Failure in development gives rise to the deformed auricle and absent external canal seen in the severer cases of congenital aural atresia. The malleus and incus are differentiated from the first branchial arch (Meckel's cartilage), and failure to differentiate results in fused ossicles. This fusion seems to be the commonest form of ossicular deformity, and occurs in a large proportion of the con-

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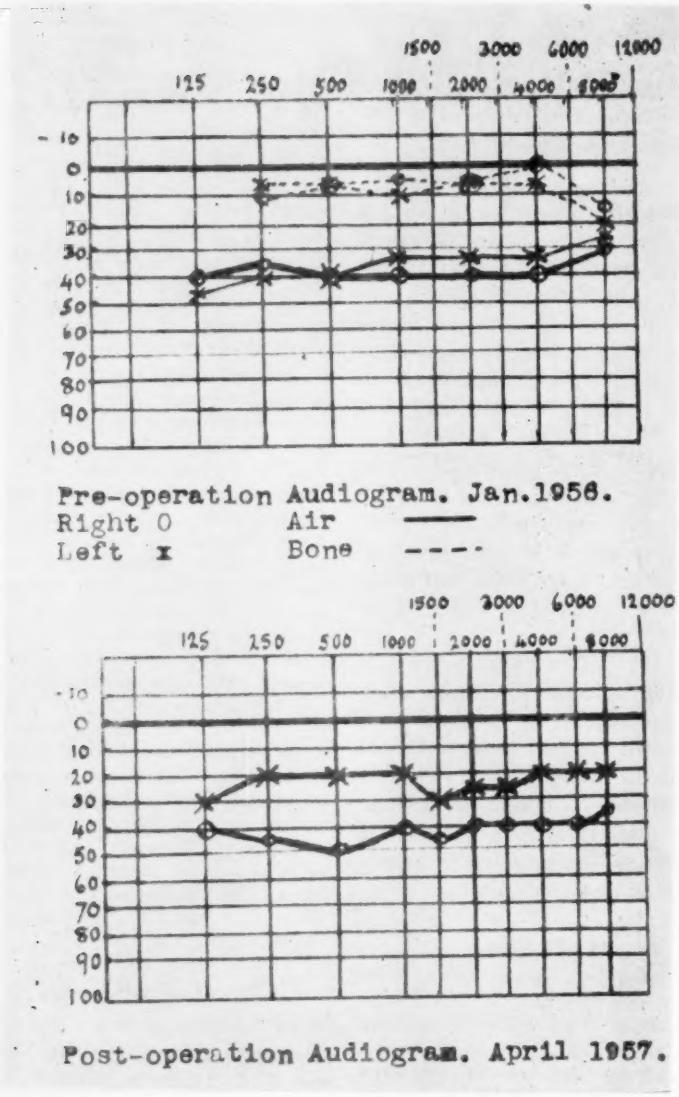

genital atresia cases. Abnormalities of the malleus alone or of the incus alone are most rare.

The stapes is often found to be normal in otherwise maldeveloped ears. This is due to the fact that it is derived from the second branchial arch (Reichert's cartilage) and seems to develop independently from the other ossicles.

Review of the Literature

Although hitherto cases of incudial malformation appear but rarely in the literature, the increasing use of the Rosen approach to the middle ear will doubtless increase our records of this condition. Anderson (1956) describes one in which the incus, with the distal portion of the long process missing, resulted in a gap in the ossicular chain. The

stapes was partially fixed by adhesions. The incus and malleus head were removed and the stapes mobilized. Slight postoperative improvement in hearing resulted.

A similar case, with an interesting method of treatment, was described by Dr. Goodhill of Los Angeles in a personal communication. This concerned a man of 63 in whom the long process of the incus stopped well short of the stapedial head. An attempt was made to restore continuity of the ossicular chain by fitting one end of a very fine-bore plastic tube over the shortened process and placing the other end in contact with the stapes. The immediate result was encouraging, but sufficient time has not yet elapsed to judge the final effect on hearing.

Thorburn (1957) records a case on which bilateral tympanoplasty was performed with success in improving hearing. In one ear, damaged by otitis in childhood, the long process of the incus was found to be defective. In the other, the long process projected beyond the stapes without making contact with it—probably a dislocation following a severe head injury.

Comment

The question arises as to whether the case here described is one of disease or a congenital malformation. The first possibility considered was that, in view of the slight stapes fixation, this might be otosclerosis associated with absorption of the ossicles. According to Simson Hall, however, absorption of both ossicles by an otosclerotic process is extremely unlikely, as this would be completely contrary to all knowledge and theory of the disease.

The long process of the incus might be absorbed by a dry necrosis or destroyed by long-standing middle-ear suppuration, but in these cases there is usually other evidence of tympanic damage. Henner and Buckingham (1956) observe that minor abnormalities of the ossicular chain, especially in children, are often found in association with a small attic cholesteatoma. It is their opinion that in this type of case both the ossicular deformity and the cholesteatoma have a congenital origin. Of four cases reported, three had synostosis of incus and malleus as the chief cause of hearing loss.

In the case reported here, there was no evidence of cholesteatoma, and no history of past earache or discharge. The drumheads appeared normal, with no sign of gross retraction, scarring, or healed perforations. These facts, together with the bilateral nature of the deformity all suggest a congenital malformation.

Summary

A case of bilateral congenital malformation of incus is described. The condition was discovered on opening the middle ear in the performance of Rosen's stapes mobilization operation.

Permission to publish this case was given by Dr. Samuel Rosen, who also gave instruction, help, and encouragement.

11 Sandyford Place (3).

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Chemodectoma or Tumor of the Glomus Jugulare and Tympanic Bodies

F. C. W. CAPPS, F.R.C.S., London

I have been fortunate since first I drew attention to glomus jugulare tumors in my country in 1952 to have collected quite a considerable series (23 cases) and to have seen or had close follow-up information about six others by the courtesy of colleagues.

In this brief paper I have endeavored to show in tabular form the results of our experience of clinical types and methods of treatment.

TABLE 1.—*Summary of Case Material*

29 Cases	26 Women	3 Men

Average age when first seen 45.5 yr.
Youngest 17 yr. Oldest 74 yr.
Some previous history known in 23 cases
Average length of such history 7.6 yr.
Shortest 1 yr. Longest 30 yr.

The great preponderance of women is well shown in Table 1.

The length of history in a high proportion of cases emphasizes the very slow rate of progress in these tumors. Table 2 gives an indication of the symptoms complained of or signs found.

Two cases were associated with carotid body tumors, one on the same, the other on the contralateral side.

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Three of the patients with intratympanic tumors did not complain of deafness and were only slightly deaf on examination.

Two cases complained of facial twitching but had no paralysis.

In one case the presenting symptom was dysphagia.

In none of the cases was there any evidence of metastasis or invasion and spread by the jugular vein.

These tumors, in my opinion, fall into two distinct classes: (1) those arising in the tympanum from bodies described by Guild as arising in the osseous canal of the branch of the 9th nerve (one-fifth), the mucosa covering the promontory (one-fourth), and some along the course of the auricular branch of the 10th nerve; (2) those, somewhat more than half, arising in the adventitia of the dome of the jugular bulb.

The former tend to polypus formation in the meatus, with tinnitus and deafness as early symptoms and facial paralysis and vertigo late.

The second group tend to spread on the undersurface of the petrous, often with considerable bone destruction. The 9th, 10th, and 11th nerves lying proximal to the vein are usually the earliest involved, then the 12th nerve, and finally, if the tip is reached, intracranial spread through the foramen lacerum may involve the 5th and 6th nerves.

TABLE 2.—*Symptoms and Signs*

Symptoms & Signs	Deafness	Tinnitus & Throbbing	Discomfort or Pain	Suggesting Acute or Chronic Suppuration	Granulation or Polyp in Meatus or Mastoid Cavity	Bleeding	Vertigo	Nerve Palsies	Intra-tympanic Swelling
Previous	12	10	3	6	3	5	2	2	4
Late			1	1	7			7	
After recurrence			1		4	2	3	2	

TABLE 3.—*Treatment and End-Result, Tympanic Cases*

Cases	Primary	Secondary	After Recurrence	End-Result
1	Polypectomy			Still under observation 1 yr.
2	Polypectomy		Tympanomastoidectomy	Alive and well 3 yr. (10); 3 yr. (20)
2	Tympanomastoidectomy			Alive & well 3 yr. (4); 3 yr. (40)
4	Tympanomastoidectomy		X-irradiation after 1, 7, 7, 7 yr.	Alive & well 2 yr. (5); 3 yr. (4); 3 yr. (10); 7 yr. (23)*
2	Tympanomastoidectomy	2d case Radium tube 300 mg. hr. to cavity	X-irradiation and later morcellement	Alive & well 1 yr. (5+); 15 yr. (18)*
1	Polypectomy, 3 times		X-irradiation	Alive & well 5 yr. (14); polyp scarred down
1	Irradiation	Tympanomastoidectomy		Alive & well 6 yr. (9); scarred down
2	Irradiation			Alive & well 1 yr. (intratympanic case; tinnitus better; hearing good) (6); 1 yr. (15); polyp with facial palsy
15				

Figures in brackets in last column=total history in years.
* Dead bone in cavity.

The tympanum is often involved late, and with it the facial nerve and labyrinth.

Polypectomy alone is almost always followed by recurrence, and, while, as indicated in one case, the third recurrence was completely fibrosed by x-irradiation, my own feeling is in favor of a tympanomastoidectomy and removal of as much tumor as bleeding and possible exposure of the facial nerve will allow. Anything residual then seems to scar down well and may not show recurrence, if any, for many years. In one intratympanic case the hearing was so good that we relied on clinical diagnosis only and treated the tumor by x-irradiation of high voltage without confirmatory biopsy. Her tinnitus has ceased; the condition looks quiescent, and she can perform Valsalva's maneuver. In another patient with polypus of 14 years' duration, who finally developed facial palsy, her age (74) and frailty prompted us to try irradiation only. The polypus is shrinking, and the facial palsy recovering after six months.

In two cases of recurrence in the mastoid cavity irradiation failed to prevent recrudescence, and in these I have, under magnification, removed the growth from the floor and promontory by morcellement. In one of these I was greatly helped by hypotension during anesthesia, though this may well not be a safe proceeding in some elderly patients.

In two patients we have, several years after irradiation of the mastoid cavity, areas of dead bone which show no sign of separating. These both involve the floor of the deep meatus and anterior surface of the facial spur. Apart from needing rather more frequent toilet, they have no disability.

Tomographic x-rays will often show jugular foramen enlargement in coronal section very well and are more useful for comparison if the lesion is thought to be progressing.

With one exception (A) all these cases had irradiation. This case which had an intratympanic tumor, proved by paracentesis and biopsy, was dealt with by the radical operation suggested by Lündgren and the final destruction of the bulb tumor by diathermy. Even in this simple case it was a

TABLE 4.—*Fourteen Glomus Jugulare Body Cases*

Fourteen Glomus Jugulare Body Cases
A. 1, Ear symptoms; intratympanic tumor seen.
B. 1, Carotid body tumor; 6 yr. later polyp in same ear; 7 yr. later, facial spasm.
C. 1, Hemangioma of ear removed; 7 yr. later recurrence and nerve palsies
D. 6, Primary paralysis cranial nerves; varying from 6—12 (9, 10, 11, 12, most frequent)
E. 5, Had operation on ear for conditions suggesting suppuration; tumor not suspected until recurrence in cavity caused nerve palsies or suggested a neoplasm

TABLE 5.—*Treatment and Results in Jugulare Body Cases*

A. Operative exposure jugulare bulb; diathermy; patient alive and well 7 yr.
B. Operation carotid body; hemiplegia; polyp removed; irradiation; alive and with some incapacity 14 yr.
C. Irradiation of recurrence; nerve palsies (except 7) recovered; died, age 75, of senility and pneumonia
D. All 6 had irradiation with improvement and recovery of some or all nerves; 5 alive and well 6, 5, 5, 2, and 1 yr.; 1 improved but still has pain and (?) needs further treatment
E. 4 had irradiation; all alive and well with varying degrees of recovery of nerves (2, 4, 5, 8 yr.); one had craniotomy and deep x-ray; died 7 yr. later of infection

formidable procedure, and I vowed that I would never undertake it for an extensive tumor with nerve palsies. The patient had postoperative palsies of the 7th, 10th, and 11th nerves and a very stormy convalescence with considerable mental disturbance. She made a complete recovery and has shown no sign of recurrence seven years after.

The rationale of irradiation is now, I think, established. The actual tumor cells are obviously radioresistant, but the great vascularity of the tumor suggests that great reduction in size and strangulation of the cells may be produced by causing an endarteritis of the vessels and blood spaces. To do this it is reckoned that 600 r per field is essential, and to cover an average tumor a minimum of 2500 r would be necessary. We have usually aimed at 4000-5000 r with a high voltage machine (1000 kv.). With the shorter wavelength which this generates the risk of radionecrosis of bone is greatly diminished. This may be getting near a dose where changes in cerebral vessels and radionecrosis of the brain might be a definite risk. Except in one case, where we suspected pituitary damage, this has not occurred, and this case recovered well without the need for hydrocortisone therapy.

It is interesting to note that in discussion of a paper on this subject which I presented to the last Congress several American critics doubted the efficacy of irradiation, as the literature stated that both these and the analogous carotid body tumors were radioresistant, but a recent study of 13 cases by Williams, Childs, Parkhill, and Pugh of the Mayo Clinic (1955) concludes:

> We believe that it is fair to conclude that radiation therapy has a definite therapeutic effect on chemodectomas of the glomus jugulare. The results seem to indicate that if surgical procedures are used, they should always be combined with radiation therapy.
>
> While too little time has passed in most of the patients who were treated with radiation therapy alone to come to a definite conclusion that this alone is to be preferred in most patients with chemodectoma there are indications that in time this will be the final conclusion.

Case B was a dramatic illustration of the dictum of Le Compte when warning against surgery of carotid body tumors "that one in three die of the operation and of those that survive eight in ten are left with a temporary or permanent neurological disturbance."

In 1952 Winship et al. gave a mortality for a collected series of 21.5%. Most of these deaths, however, were due to operative measures or occurred because the tumors were too far advanced for any treatment to be of avail.

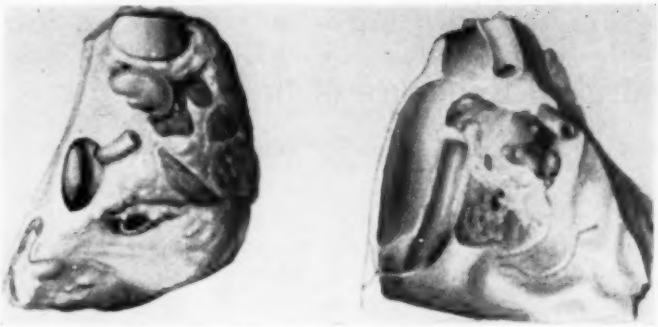
Two of the cases I have here reported are dead, one at the age of 75 of bronchopneumonia, cerebral thrombosis, and arteriosclerosis, who had had no sign of active tumor for four years, and the other who died seven years after a craniotomy and irradiation from a spreading infection after cellulitis of the pinna.

At least half the cases I have reported have a combined history and personal observation ranging from 25 to 10 years and it would seem that, although we cannot ensure destruction of these tumors, we can, by judicious operation, radiation therapy, or a combination of the two, keep these cases under control.

Our experience has been that in suitable cases (the tympanic group) operation may give long-lasting cure, and one can safely reserve radiotherapy until such time as recurrence is obvious. The effect of postoperative scarring, given time, may be entirely adequate, while immediate radiation in a raw or recently healed area may cause delayed healing, bone necrosis, and a long-standing messy cavity.

May I in conclusion show you a photograph of a postmortem specimen obtained by Mr. Savage of Chelmsford. This lady, aged 49, was being sent to see me, but died suddenly of a cerebral hemorrhage within a week of the appointment being made. Mr. Savage wrote:

> Patient came to see me complaining of two to four years right tinnitus and deafness. She has not troubled about this but five to six weeks before



Specimen of glomus jugulare tumor.

I saw her she started "giddy turns." In these she would fall to the right and vomit. Three years ago she had some neuralgia on the right side of her face in attacks lasting up to two hours.

I found an interesting appearance of the right tympanic membrane strongly suggestive of a glomus jugulare tumour. There was a bulge postero-inferiorly but a kidney shaped violaceous swelling was visible over a much wider area posteriorly and inferiorly. The drum was intact. She had what appeared to me to be a right perceptive deafness of great severity so that she could only just hear a conversational voice at the ear. Bone conduction was much reduced and Weber lateralised to the left. This was confirmed by an audiogram. The only X-ray abnormality, if indeed it is an abnormality, is an enlarged foramen spinosum on the right side. Dr. Buckley, our Neurologist, does not find any evidence of intracranial involvement.

At post-mortem the glomus tumour was found as anticipated but was much larger than had been thought clinically. It occupied most of the inner ear and also filled the lateral sinus and jugular bulb and protruded into the tympanic cavity.

16 Park Sq. E., Regent's Park, N. W. 1.

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Surgical Exposure of the Facial Nerve

STEPHEN P. HOGG, M.D., and ROBERT C. KRATZ, M.D., Cincinnati

A complete knowledge of the anatomical relationships of the facial nerve is necessary for safe and effective surgery of the temporal bone, parotid gland, and other surgical problems in the upper neck and posterior mandibular area, as well as surgical exposure of the nerve itself.

After the surgeon has positively identified the facial nerve he proceeds with safety and greater effectiveness in these anatomical areas.

A very precise and constantly applicable procedure for promptly isolating the facial nerve is needed. Although this problem is an old one, surgical dissection in this area is still uncertain for many, and unfortunately at times the facial nerve is injured. The gravity of a facial nerve paralysis does not require any elaboration.

I should like to describe briefly a surgical approach which consistently identifies the facial nerve. The reason this method is precise and constant is because it refers to constant anatomical landmarks.

Repeated anatomical and surgical dissections reveal a constant relationship between the tympanomastoid fissure and the facial nerve. This is the suture line between the mastoid and tympanic portions of the temporal bone. The facial nerve is located 6 to 8 mm. medial to this readily identifiable landmark.

A postauricular incision beginning 1 cm. inferior to the superior attachment of the auricle and extending as far inferiorly along the inferior margin of the mandible as necessary to expose the pathology is the most satisfactory incision. This incision may be extended anterior to the auricle if necessary. However, adequate mobilization and exposure can be accomplished without this extension unless some special problem exists.

The postauricular incision is carried through the periosteum of the mastoid portion of the temporal bone. The soft tissues, periosteum, and external auditory canal are retracted anteriorly, revealing the tympanomastoid fissure. The facial nerve is a large structure 6 to 8 mm. deep or medial to this point. Elevating the soft tissues and retracting them anteriorly permits prompt identification of the nerve (Figs. 1 and 2).

Fig. 1.—Demonstration of exposure of the right facial nerve with the capsule of the parotid gland intact and retracted anteriorly. This shows the relationship of the facial nerve to the tympanomastoid fissure of the temporal bone. The sternocleidomastoid muscle, digastric muscle, and styloid process with muscles attached are also visible.

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From the Department of Otolaryngology, College of Medicine, University of Cincinnati.

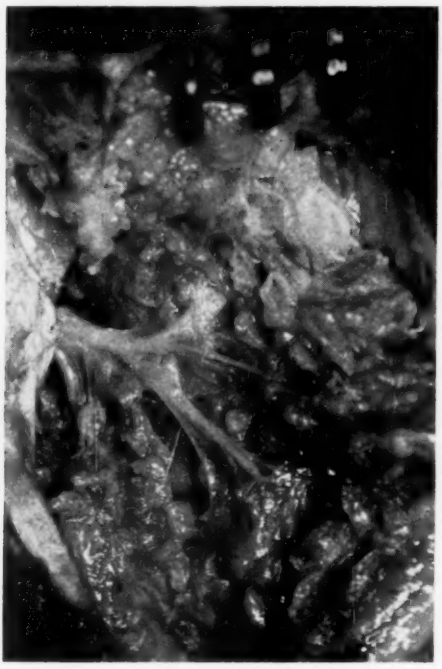

Fig. 2.—Shows the right facial nerve emerging just medial to the tympanomastoid fissure and proceeding through the parotid gland. The nerve is first identified with the parotid fascia intact, using the tympanomastoid fissure as a guide.

Although the stylomastoid foramen is 1 cm. medial to the inferior lateral margin of the tympanomastoid fissure, the nerve is encountered at a depth of only 6 to 8 mm. since it courses immediately lateral and anterior in order to pass lateral to the styloid process.

The tympanomastoid fissure guides us to the stylomastoid foramen and facial nerve.

Additional findings based on repeated anatomical dissections that deserve special emphasis are as follows:

1. Maintaining the mandible in closed position opens the retromandibular area. This opens the operative field and makes the surgery much easier.

2. The incision should be placed close to the auricle, thereby avoiding the insertion of the sternocleidomastoid muscle.

3. Henle's spine, or the suprameatal spine, located posterosuperior to the bony external auditory canal, may serve as an additional landmark for some temporal bone surgeons more accustomed to operating in this area. The tympanomastoid fissure is located directly inferior to the suprameatal spine.

4. Stay posterior to the capsule of the parotid gland. Do not enter the gland. The facial nerve emerges from the stylomastoid foramen and proceeds immediately into the parotid gland. Retracting the entire parotid gland with its capsule intact establishes the plane of dissection.

5. There is an obvious hazard in using the styloid process as a landmark for identifying the facial nerve. Since the nerve crosses lateral or superficial to the styloid process, when you reach the styloid process you are deep or medial to the nerve, possibly passing through the nerve en route.

Recently published recommendations directing attention to the mastoid process in locating the facial nerve were obviously based on the effectiveness of this general approach; however, a more specific location of the facial nerve as demonstrated in reference to the tympanomastoid fissure offers more than an elaboration on a general approach.[1]

These findings show a precise method for an operation that requires precision for a consistently safe outcome. Confining the location of the facial to this small, constantly occurring anatomical area should increase the effectiveness of surgeons in dealing with problems in this area. The surgical attack on problems in this area becomes effective only after the surgeon has the facial nerve safely identified.

The Doctors' Building.

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Submaxillary Gland Surgery and Its Complications

GEORGE E. LIEBERMAN, M.D., Philadelphia

With all the talk that is going on about the new fields open to the otolaryngologist, it would be well to examine carefully a small segment of one area. I refer to external surgery of the submaxillary gland, in its relation to the over-all picture of radical neck surgery.

There is, of course, no substitute for cadaver surgery in a proper preparation for doing a good and safe submaxillary gland resection. If the cadaver neck is dry, hard, and atrophic, an injection of 30 or 40 cc. of plain tap water into the tissues will soften the skin, the subcutaneous tissue, and the muscles, so as to simulate conditions in the living. Twenty to twenty-five complete operations on the cadaver should be a minimum, in preparation for a confident and competent approach to the actual patient. Just as in fenestration and stapes mobilization preparation, there can be no short cuts.

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Read before the combined meeting of the Philadelphia Laryngological Society and the Section on Otolaryngology of the College of Physicians of Philadelphia, Dec. 3, 1957.

At this time, a brief review of the anatomical points of the submaxillary gland, and its exact position in the neck might be in order. The submaxillary gland, when not inflamed or engorged, is irregular in form and about the size of a walnut. A considerable part of it is situated in the submaxillary triangle, reaching forward to the anterior belly of the digastricus and backward to the stylomandibular ligament, which intervenes between it and the parotid gland. The submaxillary gland has two main parts on its superior surface, an upper and a lower part. The upper part lies against the submaxillary depression on the inner surface of the body of the mandible, and the lower part is directed downward and outward and is covered by skin, platysma, and deep cervical fascia. It is crossed by the anterior facial vein and by filaments of the facial nerve.

The submaxillary duct, or Wharton's duct, is about 5 cm. long and has very thin

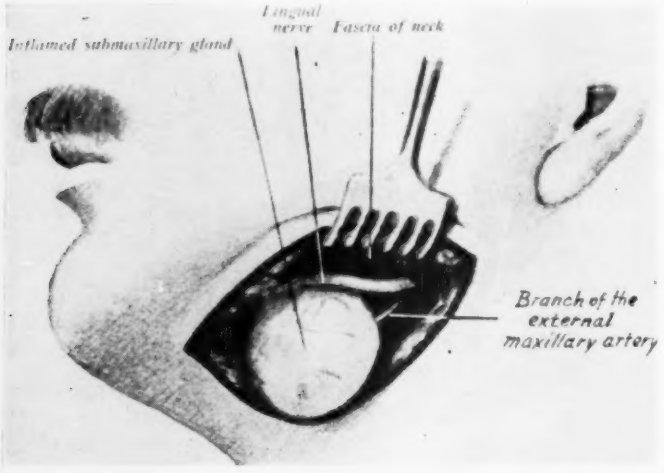

Fig. 1. — Exposure of enlarged submaxillary gland. (Reproduced with permission from Wolf, G. D.: Ear, Nose and Throat, published by J. B. Lippincott Company, Philadelphia, 1947.)

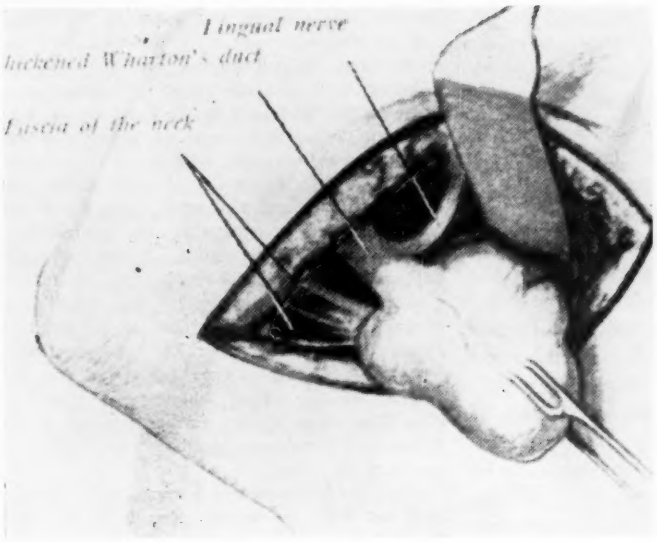

Fig. 2. — Removal of salivary gland. (Reproduced with permission from Wolf, G. D.: Ear, Nose and Throat, published by J. B. Lippincott Company, Philadelphia, 1947.)

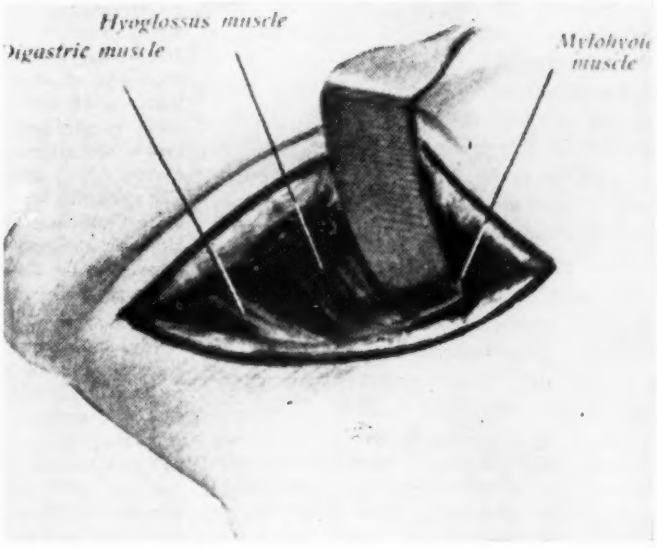

Fig. 3. — Floor of wound cavity. (Reproduced with permission from Wolf, G. D.: Ear, Nose and Throat, published by J. B. Lippincott Company, Philadelphia, 1947.)

walls. It begins by numerous branches from the under surface of the gland, goes upward and forward into the mouth, and opens by a narrow orifice on a summit of a small papilla, at the side of the frenulum linguae. The arteries supplying the submaxillary gland are branches of the external maxillary and the lingual; veins follow the arteries, and the nerves come from the submaxillary ganglion, through which it receives branches from the chorda tympani and the lingual.

The actual important surgical anatomy is relatively simple. Under general or local anesthesia, an incision 3 or 4 in. in length is made parallel to the lower jaw, from in front backwards, and about a finger's breadth below its margin. The incision should include skin, fat, platysma, and

fascia, and should expose the surface and the lower margin of the gland. All bleeding points should be tied at once. A fairly large vein, a branch of the facial, runs close to the gland and should be double tied and divided. Finger dissection, blunt dissection, or gauze dissection should be the order of the day. Soon the gland begins to lift up as on a pedicle and a branch of the facial artery is visible at the posterior end of the gland. This branch is tied, and the main vessel pushed away. Eventually the lingual nerve comes into view at the upper margin of the gland. The nerve should be gently dissected away by blunt dissection, and, if attached to the capsule, by scissor dissection.

About this time the duct is completely in view, and is tied off as high up as possible, while the assistant applies finger pressure to the floor of the mouth. After the gland is removed, the digastric, mylohyoid, and hyoglossus muscles can be seen, as well as the hypoglossal nerve.

Loose petrolatum packing with some Neosporin ointment (ointment containing polymyxin B, bacitracin, and neomycin) is used in the cavity; no deep sutures are employed, in order to prevent injury to nerves and avoid any chance of infection.

Now to the citing of several of the actual cases, and their complications. There were 17 cases in all, involving the external removal of the submaxillary gland. The years of surgery and postoperative observation were from 1946 to 1957.

Case 4.—A woman aged 52 years complained of foul-smelling pus forming underneath the tongue and of a painful swelling beneath the right mandible. The pain, pus, and swelling were worse on eating. The condition had existed for several months. X-rays taken revealed a large stone in the duct, near the body of the gland. Under general anesthesia, the entire submaxillary gland, including a large, hard stone, approximately 1 cm. in diameter was removed. Pathologic examination at the Graduate Hospital of the University of Pennsylvania revealed a large, cystic, and inflamed gland with a single, large salivary calculus.

The patient had an uneventful hospital stay of four days, and went home quite pleased. However, about three weeks after the operation, she began complaining of severe burning of the tongue on the right side. Careful examination of the wound and the mouth revealed no evidence of swelling, inflammation, or pathology of any kind. X-rays taken again showed no stone or abscess. Local treatment to the tongue in the form of procaine injections proved of no avail. A neurologist was called in to see the case, and felt that there might have been injury to the lingual nerve at the time of the operation, and that perhaps even a neuroma had formed. He recommended using vitamin B12, and nicotinamide, and waiting six months before exploring the wound. The burning of the tongue continued for two whole years, after which time further x-rays were taken, showing a long, thin calculus in the oral remains of the duct. Under local anesthesia the duct was incised, the stone removed, and the patient sent home feeling fine. This time, there was no further burning of the tongue, and the patient has been completely well for some six years.

Case 7.— A woman aged 58 complained of severe pain and tenderness in the left side of the neck for some five days. She gave a history of having diabetes for many years and of taking 20 U. of protamine zinc insulin daily for several years. Examination revealed a foul-smelling discharge from under the left side of the tongue, with a good deal of swelling and redness of the loose tissues in the floor of the mouth. Externally, the neck showed a large, tender swelling in the general area of the left submaxillary gland. An x-ray film showed a large stone in the left submaxillary duct. The stone was removed under local anesthesia, and the patient seemed to improve for several weeks. However, the swelling, pain, and tenderness soon returned in the floor of the mouth and externally in the neck, all on the left side.

Under general anesthesia, a large, swollen, bloody, and diseased left submaxillary gland was removed. But all was not well with the facial expression of the patient; the left lower lip sagged and could not be moved normally or voluntarily. Apparently, a cervical branch of the left facial had been traumatized or cut. Trauma during the operation was most likely, since the voluntary movement of the left lip improved considerably in the next 12 months. Fortunately, the woman was not young, and did not appear too distraught by the complication.

Case 10.—A man aged 35 called me one Sunday night about five years ago, complaining of terrific pain in the mouth, and under the left lower jaw. His family physician had already administered morphine, but the pain still persisted. Examination revealed a markedly swollen and tender sublingual area, as well as a large, tender swelling of the left submaxillary region. In the hospital, x-rays showed complete calcification of the entire Wharton's duct on the left side. Under general

anesthesia, we removed five large stones intraorally from the duct. The patient made an uneventful recovery and was sent home in five days.

Three months later, the patient again had a cellulitis of the floor of the mouth, and a swollen left submaxillary gland. X-rays revealed no further stone formation, but it was obvious that the gland had to be removed. We did so, and the report came back: marked chronic inflammation of the entire left submaxillary gland. The patient made a fine recovery, and all was well for two whole years. Again, the patient complained of pain and swelling in the floor of the mouth, and the x-ray showed a large stone in the remnant of the left submaxillary duct. After three days of penicillin, we were able to go in with a little local anesthesia and remove a large rough stone, about 1 cm. in diameter. There was also a great deal of pus present, and with its expulsion the patient felt instant relief. He was sent home within a week of admission to the University of Pennsylvania Hospital.

Summary and Comments

Here we have a relatively small group of cases involving submaxillary gland surgery, and yet it is possible to fall into error and be subject to annoying complications.

Certainly, the otolaryngologist with a little study on the cadaver and observation in the living can do this type of neck surgery. Care, precision, and confidence are necessary to avoid injury to the lingual nerve and to the cervical branches of the facial nerve.

2019 Pine St. (3).

The Role of Palpation in Examination of the Throat

WILLIAM O. LODGE, M.D., Halifax, England

In clinical surgery the necessity for a methodical examination is obvious. One conceives a greater regard for palpation when one considers that the labyrinth which contains the receptors for the auditory and static senses was primordially a tactile organ of ectodermal origin. Is feeling believing? Yes.

Having listened to the history and made an inspection, one proceeds to palpate the neck from above and behind the head, comparing one side with the other. Most information will be gained if the head is supported by a pillow or headrest so that the muscles are fully relaxed.

Thyroglossal cysts are not always exactly in the middle line of the neck. A swelling beside the foramen caecum of the tongue may comprise the only thyroid glandular substance that the patient possesses. Surgical emphysema from escape of air into the tissues from the respiratory tract or inflation of laryngoceles when the patient blows out the cheeks will be recognized. Abnormal pulsation or alteration in the pulse rate on pressure over a glomus tumor will be appreciated. Rarely, one may encounter eggshell crackling in the wall of a calcified cyst.

To the erudite touch, varying grades of consistency of cervical swellings are perceptible. Large metallic foreign bodies may be palpable. Malignant glandular metastases may be very hard. Salivary calculi are firmer to the touch than the cartilaginous deposits in the neck of polyotia and these than the much larger discrete glandular swellings of lymphadenoma. There are grades of fluctuation, from that of cystic hygroma to collar-stud abscesses in connection with tonsillar tuberculosis or Bezold's mastoiditis. Simple hypertrophies are usually of about the same consistency as normal tissues. Among very soft tumors are lipomas, commonly found in the supraclavicular and suprasternal regions; pinch-

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Otolaryngologist to the Hospitals of Halifax and Huddersfield.

Fig. 1.—Branchial cyst. Skin incision.

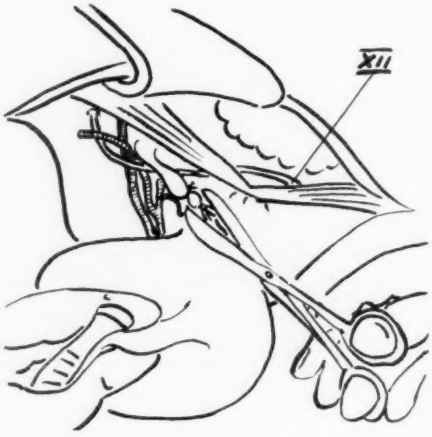

Fig. 2.—Removal of fundus of cyst to gain access to stalk.

Fig. 3.—Transposition of stalk to submaxillary triangle.

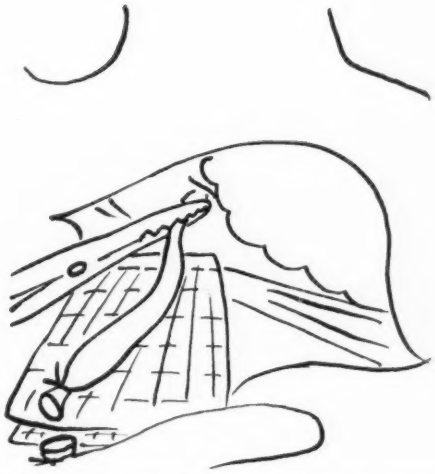

Fig. 4.—Tracing back to supratonsillar region of pharynx.

ing up the skin over them may produce typical dimpling. That swellings low in the neck may be glandular metastases from occult primary carcinoma in the thyroid gland is well recognized; aberrant thyroid tissue is relatively rare. There is another kind of swelling usually found in the vicinity of the carotid bifurcation which is exquisitely soft—the branchial cyst—in the recognition of which palpation plays such an important part that it is worthy of a paragraph to itself.

Branchial cysts usually arise from the second of the clefts between the five branchial arches which appear during the first three weeks of embryonic life. They pass out into the neck between the internal and external carotid arteries, in close relation to the caudal group of cranial nerves, as shown in the accompanying diagrams (Figs. 1 to 6). Fistulae and cysts derived from the first cleft are related to the pharyngotympanic tube and external auditory meatus. Those derived from the third cleft would pass between the common carotid and the vagus, unless they were thymogenic, in which case they would pass ventrally to the carotid. They are generally encountered in young adults. Since they

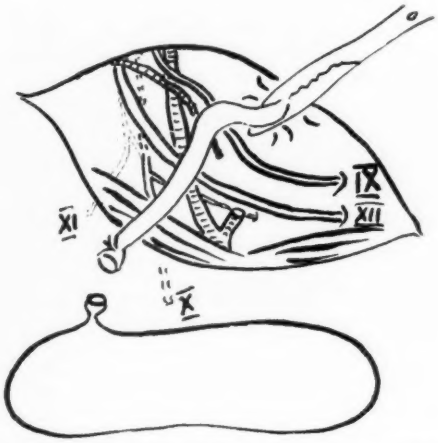

Fig. 5.—Relation of caudal group of cranial nerves to the cyst.

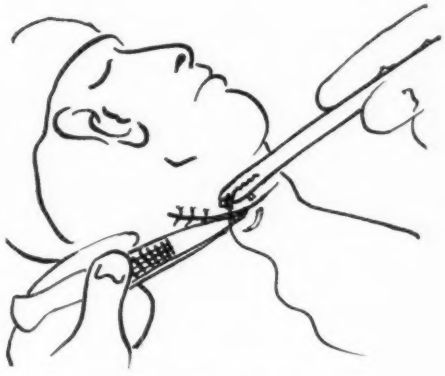

Fig. 6.—Closure of the wound.

Fig. 7.—Removal of a pleomorphic salivary adenoma from the palate—a nodular growth, encapsulated, but, where friable, gritty to the touch.

are a little more prone than normal tissues to malignant degeneration, no competent surgeon need hesitate to remove them.

With regard to palpation of the pharynx, one tends to make a more satisfactory examination if surgical gloves are available. Again, bimanual palpation is often of value. Certain landmarks such as the hamular and styloid processes are palpable rather than visible. In the nasopharynx, the finger is almost as good as the eye, though certain occult carcinomas are very difficult to detect by any method of examination.

A nodular swelling which contrasts well with the smoothness of a branchial cyst is the pleomorphic adenoma or mixed parotid tumor. The removal of a growth of this type from the palate is shown in Figure 7. Such swellings usually present themselves at a time of life when arteriosclerosis and other systemic diseases have had time to set in. They are fairly well encapsulated, but, if any portion of the tumor has to be removed piecemeal from the base of the skull, it is easily recognizable because of its gritty cartilaginous feel. Thus palpation may play an important part during the course of an operation. If mitoses are frequent, on histology, postoperative irradiation is indicated, but sensitivity is not such as to make irradiation appear preferable, from the beginning, to surgery.

Tactile sensation may be projected to the end of a probe; for example, during investigation of posterior choanal atresia, and this is perhaps the only true use of a probe—as a delicate antenna.

Always of significance is the mobility of a swelling in various planes.

Summary

The tactile qualities and varied consistency of certain swellings in the throat are discussed. The exquisite softness of branchial cysts is contrasted with the nodular hardness of pleomorphic salivary adenoma. An endeavor is made, by line drawings, to evoke these tactile sensations visually.

Successful palpation depends largely on complete relaxation by posture of the patient's muscles.

Green Leas, Savile Park.

Tonsillitis and Rheumatic Fever

LAYTON S. ROGERS, M.D., Whittier, Calif.

Through the courtesy of the Los Angeles County General Hospital a series of cases of rheumatic fever is reported here. These cases are the admissions for acute rheumatic fever for a two-year period during the fiscal years of 1954-1955 and 1955-1956. There were 97 patients admitted for this disease during the period, and the records of 87 of these patients were available for review. An analysis of these 87 records is presented here with particular reference to history of preceding respiratory infection, throat culture, and tonsillectomy.

Acute arthritis is a disease that has been known for centuries and it is reported to have been described by Hippocrates. The term rheumatism as a description for acute polyarthritis was first used in the 17th century by Guilloume de Baillon, and in 1676 Sydenham published a clinical description differentiating acute arthritis from gout. In 1788 David Pitcairn in a lecture at St. Bartholomew's stated that persons subject to rheumatism were attacked more frequently than others with symptoms of organic heart disease, and in 1789 Edward Jenner in a lecture before the Fleece Medical Society described disease of the heart following acute rheumatism. In 1843 Bouilland stated his law of coincidence. This was the coincidence of diffuse acute articular rheumatism with fever and rheumatism of the serofibrinous tissue of the heart. In 1904 Aschoff established the specificity of the heart lesion in rheumatic fever.

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From the Los Angeles County General Hospital and the Department of Otolaryngology of the University of Southern California School of Medicine.

Since the recognition of rheumatic fever as a disease separate from the other rheumatic conditions, the etiology of this disease has caused a great deal of speculation. It was early noted that there is a relationship between upper respiratory infection and the onset of rheumatic fever, and the early writers were particularly impressed with tonsillitis as being often associated with the disease. The etiology of rheumatic fever is still not entirely clear, but in the early 1930's Coburn [1] in this country and Sheldon [2] and Glover and Griffith [3] in England began bringing to our attention the fact that hemolytic streptococcus infections often preceded an attack of rheumatic fever and that in succeeding attacks this organism was often present. In 1939 Jones and Mote [4] reported on the throat culture in a series of rheumatic fever cases. Respiratory infections preceded 58% of the first attacks and 66% of these were sore throats. In recurrent attacks 67% were associated with respiratory infections. Throat cultures showed hemolytic Streptococcus in 23% of children hospitalized with rheumatic fever and 28% in children hospitalized without rheumatic fever. In the series reported here previous history of respiratory infection occurred in 49.4%, and a positive throat culture for hemolytic Streptococcus occurred in 39.4% (Table 1).

Swift [5] believes that there are transient hemolytic Streptococcus infections in the respiratory membranes which do not cause symptoms, and this accounts for the fact that many times a previous history of infection is not available.

TABLE 1.—*Previous Respiratory Infection and Throat Culture in Cases of Rheumatic Fever*

Previous upper respiratory infection	49.4%
Positive throat culture for β-hemolytic Streptococcus	39.4%
Elevated antistreptolysin-0 titer	96.4%

The Streptococcus has been the subject of a good deal of investigation. The work of Griffith [6] in England and of Lancefield [7] in this country has been responsible for the classification of the Streptococcus into at least 12 groups which can be identified serologically. These groups are characterized by a serologically active polysaccharide which is specific for each group. Of these groups, Group A is the hemolytic Streptococcus that is responsible for the many severe diseases in man. Other groups are found in man, cattle, horses, dogs, and milk. There may be nonhemolytic members in any group, but this is very rare in Group A.

Griffith [6] with special agglutination methods typed strains of hemolytic streptococci from cases of scarlatina. He first established 4 types and later 27 types. Twenty-three of these types were later found to be in Group A, three in Group C, and one in Group G. Lancefield [7] later was able to identify something over 40 specific types of Group A by the use of type-specific proteins. Protein M is the antigen primarily associated with virulence. There is also another antigen known as the T substance which produces what is called bacteriostatic antibody. This changes a virulent Streptococcus which cannot be phagocytized into an organism which can be phagocytized. These two antigens are able to form antibodies when injected into animals, and these antibodies can be identified by agglutination tests. These antigens appear to be responsible for type specificity in Group A streptococci. However, the same type is not always responsible for a single disease, and several types may cause scarlet fever or other Group A Streptococcus diseases. Also different Streptococcus diseases may be caused by a single type. In Group B the antigens responsible for type specificity are polysaccharides and are probably so in all groups except A and C.

Swift [5] describes these as the cellular components of the streptococci and then goes on to describe a group of extracellular components. These extracellular antigenic components are the products of the growth of Streptococcus in the media in which they grow and in the tissues of infected patients. These extracellular antigens are (1) two streptolysins, O and S, which are responsible for the hemolytic properties of the Streptococcus; (2) the erythrogenic toxins, which are responsible for the rash in scarlet fever; (3) fibrinolysin, which is an enzyme and dissolves the clot of normal human blood, and (4) hyaluronic acid, which is a capsular substance and is found in the interfibrillary cement substance of collagen and in many other body tissues.

Of these extracellular components, streptolysin-O and fibrinolysin, also known as streptokinase, are known to produce antibodies called antistreptolysin-O and antistreptokinase. Techniques have been devised for quantitive determinations of these antibodies, and these determinations have become a help in the diagnosis of rheumatic fever. However, all strains of Group A Streptococcus do not produce these toxins, and this accounts for the negative tests in some Group A infections. Swift [5] reports that 80% to 90% of rheumatic fever cases will show elevated antistreptolysin-O titers and points out that these are not specific tests for rheumatic fever but only show evidence of previous streptococcal infections. In the cases reported here, 96.4% had elevated antistreptolysin-O titers (Table 1). Attempts have been made to show that higher values for antistreptolysin-O titers occur in rheumatic fever patients, but this has not been completely accepted.

Hyaluronic acid, hyaluronidase, and antihyaluronidase have also been under investigation in relation to rheumatic fever. Relatively higher concentrations of antihyaluronidase have been reported in rheumatic fever patients, but Group C Streptococcus, which has no relation to rheumatic fever, Pneumococcus, and Staphylococcus produce large amounts of hyaluronidase.

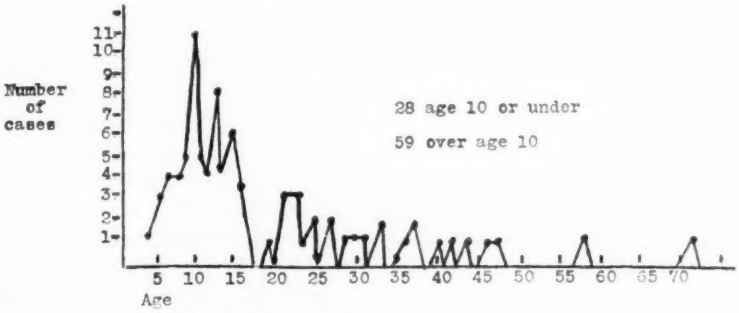
The primary pathology in rheumatic fever involves connective tissue. It is one of the collagen diseases, which include periarteritis nodosa, lupus erythematosus, rheumatoid

arthritis, generalized scleroderma, and dermatomyositis. In rheumatic fever there is a phase of exudation, as in the effusion into joint cavities, pericardial sac, and pleural cavity, and a phase of proliferation as evidenced by the heart muscle lesions and the subcutaneous nodules. The Aschoff body in the heart muscle is regarded as the specific lesion and is made up of a central area of necrotic collagen and fibrinous exudate surrounded by large multinuclear cells. There is no typical cell found in either the exudative or the proliferative lesion.

Rich [8] has emphasized the edema of the collagen fibers in the heart muscle and believes that there is evidence that the pathological picture in rheumatic fever, periarteritis nodosa, disseminating lupus erythematosus, and rheumatoid arthritis is due to the result of an anaphylactic type of hypersensitivity. Two types of hypertensitivity are described, the anaphylactic type and the tuberculin type. The anaphylactic type has a prompt reaction, an effect on smooth muscle, and causes vascular damage which interferes with cell nutrition. An example is the asthma attack. The tuberculin type is a local reaction which develops slowly, causes no effect on smooth muscle, and causes a direct effect on the cell and not on the blood vessels. An example is the skin reaction to tuberculin in skin testing for tuberculosis. Rich and Gregory [9] injected horse serum into rabbits, sensitizing them, and then with another injection created serum sickness. In some of the rabbits with serum sickness lesions developed in the heart muscle and heart valves similar to the heart lesions in rheumatic fever. It is believed that these lesions are of an anaphylactic hypersensitivity type, and several similarities in the clinical picture of serum sickness and rheumatic fever are listed. Murphy and Swift,[10] using rabbits, injected a different specific type of Group A Streptococcus each time at varying intervals over a period of several months. A few of the animals developed fatal illness. Some died of a bacteremia, and some showed at autopsy heart-muscle and valve lesions similar to those found in rheumatic fever.

The age of greatest incidence of rheumatic fever has usually been reported to be in childhood. Gibson [11] reported the highest incidence in his group of cases to be at age 6, while Rammelkamp, Wannamaker, and Denny [12] report the attack rates about the same in 29-year olds as those under 11. In the series reported here, the highest incidence was at age 10, with 28 cases age 10 or under, and 53 cases over age 10 (Figure). Gibson [11] reports the incidence slightly higher in girls, while the series reported here showed the incidence slightly higher in boys (Table 2).

Paul [13] estimates that about 3% of those persons with a Group A Streptococcus infection will develop rheumatic fever, and Rammelkamp and co-workers [12] report that between 2.4% and 3.1% of those with Group A Streptococcus infections will develop rheumatic fever. Rates appear to be about the same in different geographical loca-



Incidence of rheumatic fever according to age.

TABLE 2.—*Incidence of Rheumatic Fever According to Race and Sex*

		Number	%
Race	White	57	65.6
	Negro	30	34.4
Sex	Male	46	52.9
	Female	41	47.1

tions, but there is little doubt that the disease is milder in warm climates, and may be very difficult to diagnose. There is also little doubt that the incidence of rheumatic fever is considerably greater in families with poor home environment. Wilson and Schweitzer,[14] in a genetic analysis of 646 children from 291 families, found 43 cases of rheumatic fever when 49 were expected. The conclusion was that rheumatic fever susceptibility is inherited as a simple recessive trait.

The diagnosis of rheumatic fever is sometimes difficult, since there is no specific laboratory test for the disease. Jones[15] in 1944 reported the following diagnostic criteria:

A. Major manifestations
1. Carditis with murmurs, pericarditis, increased P-R interval, inverted T waves and changes in axis deviation
2. Arthralgia that can be related to previous Group A streptococcus infections
3. Chorea: One-half of rheumatic fever patients have chorea.
4. Subcutaneous nodules: These are rare in the early stages, and carditis is almost always present with these. They are a help in determining activity in old cases.
5. Recurrences: 70% of Jones' series had recurrences within 10 years.

B. Minor manifestations

These are fever, adbominal pain, rashes, precordial pain, epistaxis, pulmonary findings.

Two minor and one major manifestation were required for a diagnosis.

Jones[16] later modified his criteria as follows:

A. Major criteria
1. Carditis
2. Polyarthritis
3. Chorea
4. Subcutaneous nodules
5. Erythema marginatum

B. Minor criteria
1. Fever
2. Arthralgia
3. Prolonged P-R interval
4. Increased sedimentation rate, and white blood count or presence of C-reactive protein
5. Preceding Group A Streptococcus infection
6. Previous rheumatic fever or inactive rheumatic heart disease

Two major and one minor or one major and two minor manifestations in this group are required for diagnosis.

Laboratory findings are changes in the electrocardiogram, anemia, high white blood count, and elevated sedimentation rate. Adams[17] refers to acute phase reactants, which include antistreptolysin-O, which is specific for Group A Streptococcus, and the nonspecific reactants, such as mucoprotein tyrosine, C-reactive protein, hyaluronidase inhibitor, and others. These are used as a guide to the continuation of cortisone and corticotropin (ACTH) therapy. They are not specific tests for rheumatic fever. Rammelkamp and co-workers[12] report that there is some evidence that Group A Streptococcus infections producing high antistreptolysin-O titers produce more cases of rheumatic fever. They report that in 29 cases with a titer less than 125 units there were no cases of rheumatic fever. In 297 cases with a titer greater than 250 units there were 10 cases of rheumatic fever.

Griffith[18] divides the disease into four types: 1. Acute pulmonary type with sudden onset, severe polyarthritis, tachycardia, and carditis. 2. Subacute polycyclic type with repeated cyclic activity. In these cases the most marked cardiac damage is found. 3. Subacute monocyclic type. 4. Subclinical type. No acute cyclic symptoms occur, but patients are found to have rheumatic heart disease.

Griffith[18] also divides the diagnostic criteria into major and minor. The major are (1) history of previous attack; (2) carditis; (3) fever; (4) migrating arthritis, and, (5) chorea. The minor are (1) pneumonitis; (2) tachycardia; (3) subcutaneous nodules; (4) erythema; (5) epistaxis; (6) purpura; (7) weight loss; (8) abdominal pain. Particular emphasis is placed on tachycardia as a diagnostic sign and as an indication of the activity of the disease.

Saslaw [19] reports a skin test for rheumatic fever in which a 5% ointment of Trafuril (the tetrahydrofurfuryl ester of nicotinic acid) is applied to the skin. Normal persons get a hyperemia and edema of the skin, while acute rheumatic fever patients get no reaction or a blanching of the skin. The test is reported 86% correct in acute rheumatic fever cases. Chorea cases get the same reaction as the normal person.

The prognosis of rheumatic fever is that of rheumatic heart disease. In 1952 Jones and Bland [20] reported a series of 1000 cases which had been followed for 20 years. Of these cases 56% had a satisfactory outcome and 44% resulted in early death or crippling heart disease. The use of prophylactic sulfadiazine and penicillin will no doubt cut down the percentage of unsatisfactory results in the next 20 years.

The treatment of rheumatic fever is divided into the immediate treatment for the acute cases, and the preventive treatment.

The immediate treatment is rest and good general care with acetylsalicylic acid, and more recently cortisone and corticotropin. There is still some question as to whether cortisone and corticotropin are of more benefit than acetylsalicylic acid in cutting down the time of activity of the disease as measured by the sedimentation rate and the titer of the acute phase reactants. Adams [17] believes that the hormones should be used in the severe cases. Saslaw [19] states that severe reactions occur in the doses required and that the problem is still not settled. At the Los Angeles County General Hospital the acute cases receive corticotropin gel, 400 units twice a day, for one month and then gradually reduced doses for a second month. With this dosage there was one case in our series which developed symptoms of Cushing syndrome.

In preventive treatment the use of a daily dose of sulfadiazine or penicillin has become generally recommended. Rammelkamp and co-workers [12] report a series of 996 cases of tonsillitis without antibiotic treatment, in which 23 cases of rheumatic fever developed. In 978 cases of tonsillitis with penicillin treatment 1 case of rheumatic fever developed. Evans [21] reported a series of rheumatic fever cases in which 100,000 units of oral penicillin were given daily. In 71 patients receiving the penicillin there were 7 Group A Streptococcus throat cultures and 1 clinical case of Group A Streptococcus throat infection. In 242 control patients there were 33 with Group A Streptococcus throat cultures and 47 clinical cases of Group A Streptococcus throat infection.

Kuttner [22] recommends a daily dose of sulfadiazine or a daily oral dose of penicillin, or a monthly dose of repository penicillin by injection. This medication should be continued until the age of 18 or for five years after the most recent attack. It is also recommended that in order to prevent initial attacks of rheumatic fever, Group A Streptococcus throat infections and scarlet fever should be treated to maintain satisfactory penicillin blood levels for 7 to 10 days. Saslaw [19] calls attention to the fact that sulfadiazine is bacteriostatic while penicillin is bactericidal, and that some Group A streptococci have become resistant to sulfadiazine while so far none have been reported resistant to penicillin.

The question of tonsillectomy arises when preventive treatment is considered, and in the early days the opinion was definite that tonsillitis was associated with rheumatic fever and that tonsillectomy was an effective preventive of recurrences. Kaiser [23] in 1936 reported a study of 48,000 school children in Rochester, N. Y., in which he compared the incidence of rheumatic fever in those who had had tonsils removed and those who had not. The conclusion was that rheumatic fever occurs about 30% less often in children whose tonsils have been removed prior to the first attack of rheumatic fever, that tonsillectomy in cases which have already had rheumatic fever does not prevent recurrent attacks, and that tonsillectomy prior to the first attack in those children who develop rheumatic fever does not lessen the number of subsequent attacks. In a group of 597

children with rheumatic fever Kaiser [23] reports a mortality rate of 7% in those who had had tonsillectomy before the first attack, while the mortality rate in those who had not had tonsillectomy before the first attack was 13%. In cases where the tonsils were removed after the initial attack the mortality rate was 4%.

Ash,[24] in reporting an analysis of 532 cases in children, concluded that tonsillectomy had little or no effect on the course of rheumatic fever, whether done before the initial attack or at any time during the course of the disease. A lower mortality rate is reported in children who had had tonsillectomy before the initial attack of rheumatic fever, but it was thought that this was because in the nontonsillectomy group there were more boys and more Negroes, in whom the severity of the disease is reported to be greater. Also the nontonsillectomy group was made up of younger children, in whom the disease and its complications are severer. Ash also reports that in the group with tonsillectomy done before the initial attack the percentage of cases taken ill at age 2 to 3 years was 3.3%, while the percentage of nontonsillectomy cases taken ill at age 2 to 3 was 15.5%. It was thought that the low susceptibility of very young children may influence these figures, or possibly tonsillectomy at a young age may cause a certain degree of protection against rheumatic fever. Ash [24] also reports an incidence of reactions following tonsillectomy in active cases of 11.8%, and in this group there was death from carditis in three cases. With the tendency to relapse, all of these cases may not have been due to surgery, but it is advised that tonsillectomy be done only when both temperature and sedimentation rate are normal. Coburn [1] also reports cases that developed reactions when tonsillectomy was done during the active stage of the disease.

TABLE 3.—*Tonsillectomy and Rheumatic Fever*

	No.	%
Tonsils in	71	84.5
Tonsils out	13	15.4

Freud, Rook, and Brunhofer [25] report four cases of reactivation following smallpox vaccination, and Ash [24] reports one case reactivated after tooth extraction.

In the series of cases reported here one case was admitted to the hospital after having a tonsillectomy during the active stage of rheumatic fever, and in order to emphasize the danger of operating on patients during the time of activity, the following case is reported in more detail:

Present illness: The patient was an 11-year-old white boy, who, two months before admission, had had a sore throat with daily afternoon rise in temperature and night sweats. He was told that he needed tonsillectomy, and two weeks later tonsillectomy and adenoidectomy were done. After this he had persistant afternoon temperature rise to 103 F, with one chill a few days after the operation.

History: He had had scarlet fever at age 5 years.

Physical examination: The patient was a fairly well-developed thin, pale, chronically ill boy with marked cheilitis and hyperemia of the gums. There were small nodes in the neck. The heart was enlarged with the P. M. I. in the 4th i. s. almost to the axillary line. There was a split P_2 and a harsh Grade 4 apical systolic murmur transmitted into the axilla.

Laboratory results: Hgb. 11.5, WBC 17,600, 60% PMN; serum alb. 4.5 gm., serum glob. 4.00 gm. per 100 cc.; antistreptolysin-O titer greater than 800 Todd units. Sed. rate: 49, 55, 59, 32, 15, 12, 38 mm. in one hour. Throat culture: A viridans alpha Streptococcus; Neisseria catarrhalis. Urine neg.

ECG: prolonged AV conduction; first degree AV block. Chest x-ray neg.; x-ray of hands neg.

Treatment: Corticotropin gel 400 u. b. i. d. for one month, then gradually reduced doses for a second month.

Course: Gradual improvement until ECG within normal limits seven weeks after admission. Given oral penicillin, 200,000 u. daily.

Tables 1 to 6 show an analysis of the cases reported here. There is certainly no evidence that tonsillectomy had any beneficial effect in these cases, and the low percentage of those

TABLE 4.—*Recurrence of Rheumatic Fever in Relation to Tonsillectomy*

Recurrence in total number of cases	51.2%
Recurrences in nontonsillectomy cases	43.6%
Recurrence in tonsillectomy cases	61.5%

TABLE 5.—*Heart Disease in Relation to Tonsillectomy*

Heart disease in total number of cases	59.8%
Heart disease in nontonsillectomy cases	57.7%
Heart disease in tonsillectomy cases	69.2%

TABLE 6.—*Mortality Rate in Relation to Tonsillectomy*

Mortality rate in total number of cases	4.6%
Mortality rate in nontonsillectomy cases	4.2%
Mortality rate in tonsillectomy cases	7.7%

who had had their tonsils removed probably indicates the feeling today that the operation is not indicated as a routine procedure. The indication is just as it was expressed by Kinkade [26] in 1949, that is, that the indication for tonsillectomy in rheumatic fever is the same as it is anywhere else. A great many of these cases, particularly those in childhood, will suffer from repeated Group A Streptococcus infection of the tonsils, and those children should have tonsillectomy when the rheumatic fever is not active. Your attention is directed again to Ash's [24] figures in the 2-to-3-year age group which show the possibility of tonsillectomy in the younger ages causing some degree of protection against rheumatic fever (Tables 3 to 6).

Summary

A review of the literature is presented, with attention to the relation of Group A Streptococcus to the etiology of rheumatic fever. The pathology of the disease is discussed, with a review of the reports of experimental work done to reproduce the pathological picture of rheumatic fever in animals. The epidemiology and diagnosis of the disease are discussed, the difficulties and importance of diagnosis being emphasized. The treatment of the disease is discussed, including the use of sulfadiazine and penicillin over a period of time in the prevention, and a series of cases are reviewed with reference to results of tonsillectomy in the disease and with reference to the importance of doing tonsillectomy when the disease is inactive.

136 N. Friends Ave.

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Tracheal Homografts in Dogs

S. DONALD GREENBERG, M.D., Iowa City

Introduction and Aims

There is no satisfactory method to replace the entire circumference of a diseased or stenotic length of trachea with either a graft or a prosthesis. The need for such a method is evident when one considers the increasing incidence of (1) traumatic injury of the trachea resulting from automobile accidents and military actions, (2) primary and secondary neoplasms of the trachea, and (3) stenosis of the trachea following chronic infections. It was the purpose of this project to devise such a method, utilizing tracheal homografts wrapped with tantalum mesh gauze.

History

The recent advances in vascular surgery using homografts to replace diseased portions of major arteries has stirred interest in the possibility of similar procedures as applied to the trachea. Even before this current emphasis, several investigators had done basic work in studying the manner of tracheal repair.[1-7] In addition to tracheal homografts many other materials have been used for tracheal grafting, including fascial grafts, skin grafts, bone, urinary bladder mucosa, glass, wire coils, stainless steel, Vitallium, polyethylene, nylon, and gelatin sponge.[3-5,8-17]

The main problem is the contraction and stenosis which develops about these grafts and prostheses. Other pitfalls are granulation tissue formation, infection, hemorrhage, and fistulae. Gebauer[3-5] has reported success in man in replacement of a segmental portion of trachea with a skin graft supported by wire; however, he did not use sleeve or circumferential grafts. Morfit[13] has concluded that in dogs a polyethylene tube joined to the trachea by plastic cuffs is the most satisfactory prosthesis. Holle[18] reports good results in dogs using "window" grafts of nylon mesh lined by autogenous fascia lata. A recent report by Keshishian[11] gives the results of work in which tantalum and steel mesh prostheses were used in man and in dogs.

Method

Before undertaking a study of tracheal homografts it was necessary to establish the procedure to be followed for tracheal anastomosis in the dog. The dog is an eminently suited animal for this work, for its trachea is about twice the length of the trachea of man and of similar diameter.[2]

The dogs were mongrels with an average weight of 13 kg. Anesthesia was obtained by using intravenous pentobarbital (Nembutal), 30 mg/kg.

In the first dog the cervical trachea was simply severed and rejoined by interrupted 34-gauge wire sutures through the cartilaginous portion and interrupted 000 silk sutures through the membranous portion. This first animal maintained a widely patent trachea except for minute granulation tissue polyps overlying the silk sutures. The animal was killed after six and a half months. In the second animal a four-ring length of cervical trachea was severed and then anastomosed with interrupted No. 1 silk sutures placed through both the cartilaginous and the membranous portions. This animal developed an anterior fistula, and subsequently silk was not used to suture the cartilaginous portion of the trachea. Two additional tracheal autografts of four tracheal rings were done. One was wrapped by a double thickness of tantalum mesh and developed no stenosis (Fig. 1); the other was not wrapped with tantalum mesh and showed some stenosis posteriorly. Both animals were killed six months after the surgery. All the dogs showed minute granulation tissue polyps about the

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In Residency Training, Department of Otolaryngology and Maxillofacial Surgery, State University of Iowa.

Fig. 1. — Photograph showing the lumen of a four-ring tracheal autograft six months postoperatively. This autograft was supported by a double thickness of tantalum mesh. There is no stenosis.

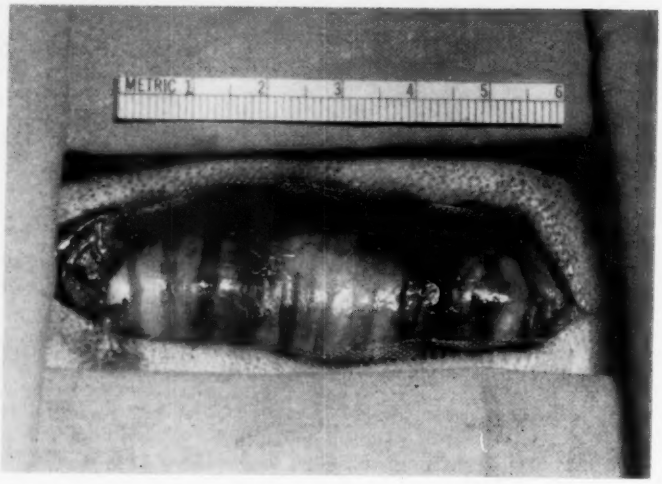

Fig. 2. — Photograph showing a five-ring tracheal homograft sutured in place. Beneath the homograft is a double thickness of tantalum mesh gauze.

interrupted silk sutures in the membranous portions of the grafts. This granulation tissue was greatly reduced after the use of a continuous row of 000 absorbable surgical sutures to close the membranous portion.

In the control group of four dogs, a vertical neck incision was made, and the strap muscles were separated in the midline. The cervical trachea was dissected free of the adjacent tissues for a length of 9 to 10 tracheal rings (approximately 4-5 cm.). The exposed trachea was severed above and below for varying lengths of three to seven tracheal rings (approximately 2-4 cm.). Retraction of the proximal and distal tracheal segments was prevented by two anterior stay sutures of 34-gauge wire. The resected portion of trachea was placed in a saline-moistened sponge and used as a homograft for a second dog, which was similarly prepared. The homograft was sutured into place, using interrupted 34-gauge wire sutures for the anterior cartilaginous portion and a continuous row of 000 plain absorbable surgical sutures for the posterior membranous portion. The upper anastomosis was completed first, allowing respiratory exchange through the distal end of the severed trachea. The tracheal sutures were through and through, and attempt was made to closely approximate mucosa to mucosa.

In a second group of 10 dogs the homografts were supported by a double-thickness wrapping of tantalum mesh gauze (0.003 in. diameter and 50×50 mesh), which was placed in the bed of the homograft before the lower anastomosis was begun (Fig. 2). The tantalum mesh was snugly

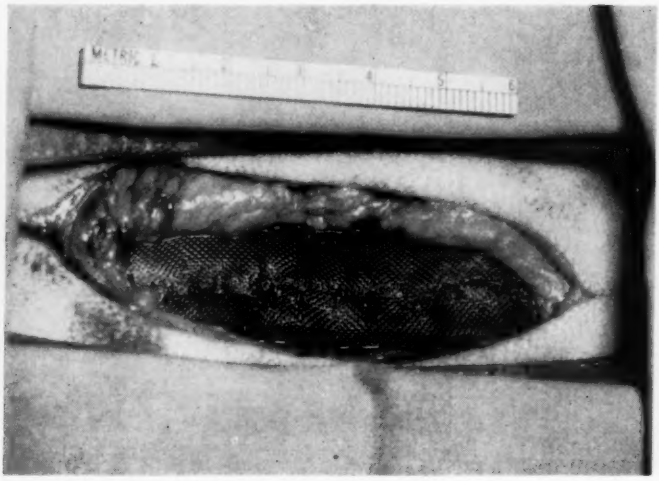

Fig. 3. — Photograph showing the double thickness of supporting tantalum mesh gauze snugly approximated about the graft and the host's trachea.

applied about the graft and extended above and below the graft for approximately two tracheal rings in either direction. The opposing edges of the tantalum mesh were approximated, using 36-gauge wire sutures, which also passed through the anterior wall of the underlying graft and trachea (Fig. 3).

The wound was closed in three layers—strap muscles, deep fascia and platysma, and skin. During the first 10 postoperative days the dogs were given daily intramuscular injections of 400,000 units of penicillin and 0.5 gm. of streptomycin.

At varying intervals the animals were followed by periodic endoscopy and tracheograms. After the dogs were killed, histologic study of the grafts was made.

Results

The control group of dogs with no tantalum mesh gauze support for the homograft averaged five weeks after grafting before they died from asphyxia because of stenosis and collapse of the trachea at the site of the homograft. The survival time of the control group after surgery was inversely related to the length of the homograft. With a homograft of three tracheal rings (approximately 2 cm.) a dog was maintained for six weeks postoperatively, while with a homograft of seven tracheal rings (approximately 4 cm.) a dog was maintained for only three weeks postoperatively. Postmortem study of these tracheal grafts showed severe stenosis and contraction, with the lining membrane formed by hyperemic granulation tissue (Fig. 4).

In the group of dogs that had tantalum wrapping the length of the homograft was varied from three to seven tracheal rings (approximately 2-4 cm.). The tantalum wrapping was double-thickness mesh gauze (0.003 in. diameter and 50×50 mesh). The majority of the shorter homografts of three to five tracheal rings (approximately 2-3 cm.) developed some stenosis approximately six months postoperatively, while the majority of the longer homografts of seven tracheal rings (approximately 4 cm.) showed stenosis from three to four months after surgery. One animal with a five-ring tracheal homograft is presently in the 13th postoperative month and as yet shows no significant stenosis. Another dog with a five-ring tracheal homograft showed no significant stenosis when it was killed six months postoperatively (Fig. 5). A third dog with a seven-ring tracheal homograft showed a satisfactory result when killed three months postoperatively (Fig. 6). With the stenosis, buckling of the tantalum mesh was seen, particularly in the longer grafts (Figs. 7, 8).

Several animals were given a two-week course of cortisone following the tracheal

Results of Study

Dog	Wt., Kg.	Graft	Length of Graft	Support for Graft	Duration	Lining of Graft	Cause of Death	Comments
1	11.5	None: anastomosis only	----	----	6½ mo.	Respiratory epithelium	Killed	Minute granulation tissue polyp posteriorly, overlying silk suture
2	13.5	Autograft	4 Tracheal rings	None	1 mo.	Granulation tissue	Stenosis	Silk alone used in anastomosis; developed anterior air fistula with intensive inflammation
3	9	Autograft	4 Tracheal rings	None	6 mo.	Columnar epithelium	Killed	Some stenosis developed after 1 mo., granulation polyp overlying silk suture
4	12.5	Autograft	4 Tracheal rings	Double layer, tantalum mesh	6 mo.	Respiratory epithelium	Killed	Satisfactory result; granulation polyp overlying silk suture
5	11	Homograft	3 Tracheal rings	Double layer tantalum mesh	8½ mo.	Granulation tissue	Intravenous anesthesia	Some stenosis appeared at 5 mo.; had tracheal dilatations during last 2 mo.
6	11	Homograft	3 Tracheal rings	None	1½ mo.	Granulation tissue	Stenosis	Stenosis appeared at 6 wk.; received cortisone postoperatively
7	14	Homograft	4 Tracheal rings	Double layer tantalum mesh	6 mo.	Stratified squamous epithelium	Intravenous anesthesia	Stenosis first appeared at 5 mo.; received cortisone postoperatively
8	14	Homograft	4 Tracheal rings	Double layer tantalum mesh	3 mo.	Stratified squamous epithelium	Stenosis	Received cortisone postoperatively and developed infection; stenosis appeared at 2 mo.
9	14.5	Homograft	5 Tracheal rings	Double layer tantalum mesh	13th mo.	----	----	Presently in 13th postoperative mo. with no significant stenosis; doing very well
10	14	Homograft	5 Tracheal rings	Double layer tantalum mesh	6 mo.	Columnar epithelium	Killed	Satisfactory result; no significant stenosis
11	14.5	Homograft	5 Tracheal rings	None	3 wk.	Granulation tissue	Stenosis	Collapse of graft which occluded lumen
12	12	Homograft	5 Tracheal rings	Double layer tantalum mesh	5 mo.	Stratified squamous epithelium	Killed	Stenosis at 5 mo.; had chronic infection of graft
14	18.5	Homograft	7 Tracheal rings	None	3 wk.	Granulation tissue	Stenosis	Collapse of graft which occluded lumen
15	13	Homograft	6 Tracheal rings	None	1½ mo.	Granulation tissue	Stenosis	Collapse of graft which occluded lumen
16	12.5	Homograft	7 Tracheal rings	Double layer tantalum mesh	3 mo.	Columnar epithelium	Killed	Satisfactory result; minimal buckling of exposed portion of tantalum mesh
17	13.5	Homograft	7 Tracheal rings	Double layer tantalum mesh	2 mo.	Granulation tissue	Stenosis	Some stenosis appeared at 6 wk.
18	15	Homograft	7 Tracheal rings	Double layer tantalum mesh	2 mo.	Granulation tissue	Killed	Buckling of the tantalum mesh was seen at 8 wk.

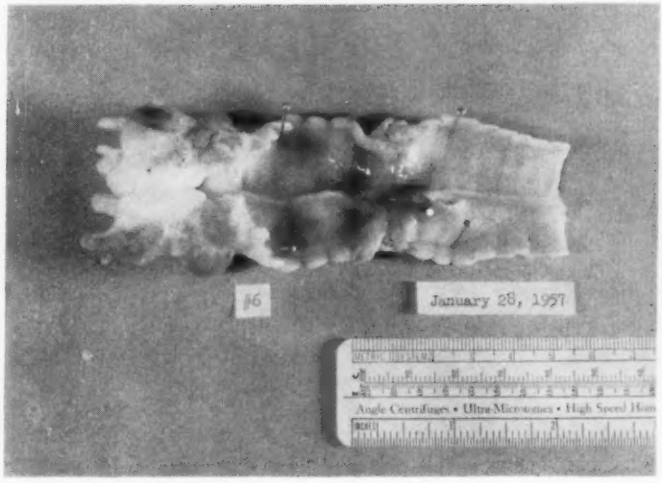

Fig. 4. — Photograph showing the lumen of a three-ring tracheal homograft. The dog died of asphyxia six weeks postoperatively. This homograft was not supported by tantalum mesh. Note the marked contraction and constriction of the homograft.

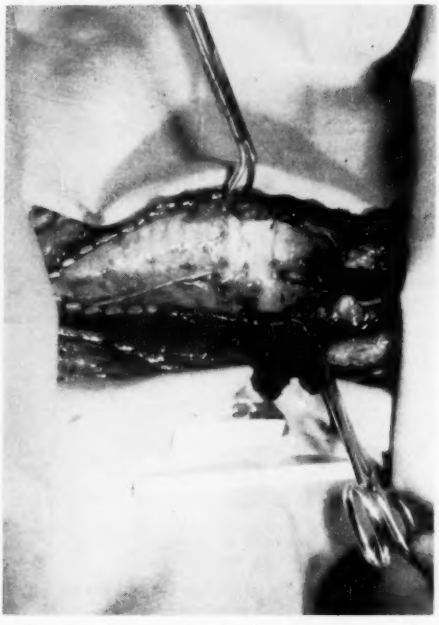

Fig. 5.—Photograph showing the lumen of a five-ring tantalum-supported tracheal homograft. The animal was killed six months postoperatively. There is no significant stenosis.

transplant. The cortisone had no appreciable effect on the over-all progress of the graft. The only case of frank infection of the homograft was in one of the animals receiving cortisone.

Several dogs had the stenosis of the graft dilated at weekly intervals for periods of two months. The results of the dilations were not long-lasting even though the trachea was dilated to a No. 30 tracheal dilator.

It was difficult to prepare satisfactory tissue sections of the homografts, for the tantalum mesh first had to be dissected free. Often the lining epithelium was thus distorted and showed artifacts. At varying postoperative periods the homografts were lined by granulation tissue, stratified squamous epithelium, and columnar epithelium. The granulation tissue was present from four to six weeks postoperatively and was associated with the acute inflammatory reaction produced by the lysis of the homograft. From approximately two to four months postoperatively, as the inflammatory reaction entered the subacute phase, stratified squamous epithelium replaced the granulation tissue as the lining membrane. Then from approximately three to six months postoperatively, after all the inflammatory reaction had completely resolved into dense fibrous connective tissue (Fig. 9), tall columnar epithelium lined the homografts (Fig. 10). The stratified squamous epithelium and the tall columnar epithelium originated from the adjacent respiratory epithelial linings of the hosts' tracheas. Respiratory epithelium, pseudo-

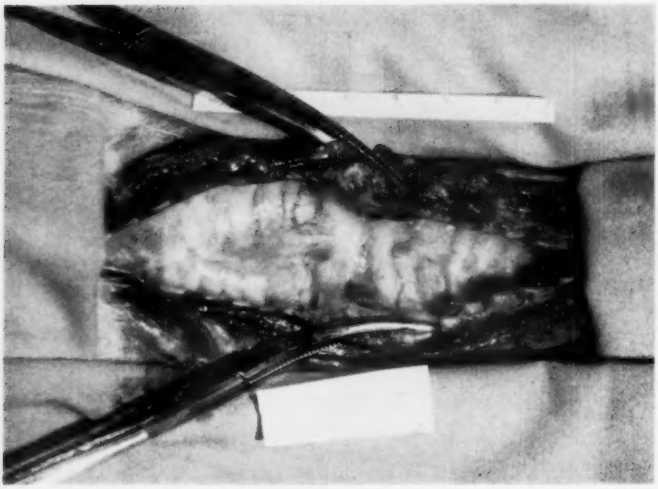

Fig. 6. — Photograph of the lumen of a seven-ring tantalum-supported tracheal homograft. The animal was killed three months postoperatively. There is no significant stenosis.

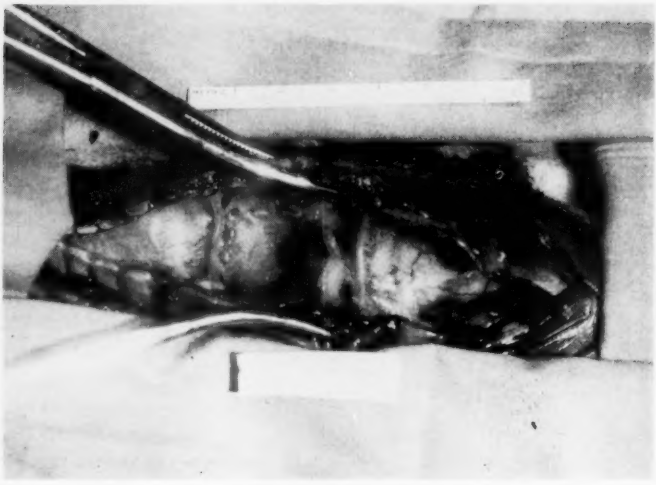

Fig. 7. — Photograph of the lumen of a seven-ring tracheal homograft. The animal was killed two months postoperatively. Note the buckling of the tantalum mesh in the central portion of the homograft.

stratified columnar ciliated, did not line the homografts. In autografts the respiratory epithelium has been seen to regenerate from columnar epithelium.[19]

Comment

The multitude of varied attempts at tracheal grafting are reminders that a truly satisfactory method has not been developed. In reviewing the pertinent literature it was found that a treacheal homograft would function well for a one-month period.[6] Such homografts lacked structural support, and they collapsed after the cartilage of the homograft was reabsorbed. It was thought that tantalum mesh gauze, which is strong and inert, would be ideal to furnish the support needed. The only difficulty encountered in using the tantalum mesh gauze has been that even when doubly wrapped it has not been of sufficient strength to prevent the constriction and contraction of the fibrous connective tissue. This was standard size tantalum mesh made of wire measuring 0.003 in. in diameter and woven to a 50×50 mesh. In the future a single layer of specially prepared stronger tantalum mesh

Fig. 8.—Tracheogram of the same trachea as shown in Figure 7. Again note the buckling of the tantalum mesh.

Fig. 9.—Photomicrograph (reduced about 43% from mag. × 100) showing lining epithelium and underlying dense fibrous connective tissue of a five-ring tracheal homograft. This tissue has been dissected free from the overlying tantalum mesh. The dog was killed six months postoperatively (see Fig. 5).

made of wire measuring 0.009 in. in diameter will be used (Fig. 11). The buckling of the double-thickness tantalum wrap is proof of the force of the newly formed fibrous connective tissue. It is significant that most of the buckling of the tantalum mesh took place toward the central portion of the graft and that the longer the homograft the more prominent the buckling and the sooner the onset of the stenosis.

Naturally the tracheal homograft will not retain its viability within the host. However,

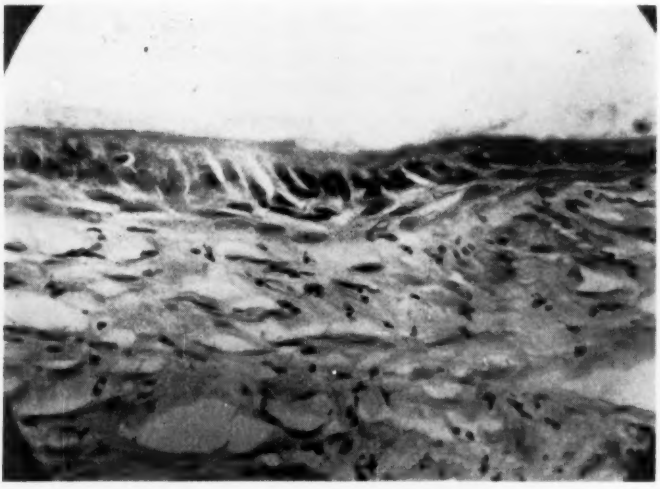

Fig. 10.—Photomicrograph (reduced about 38% from mag. × 600) showing in greater magnification the columnar epithelium pictured in Figure 9.

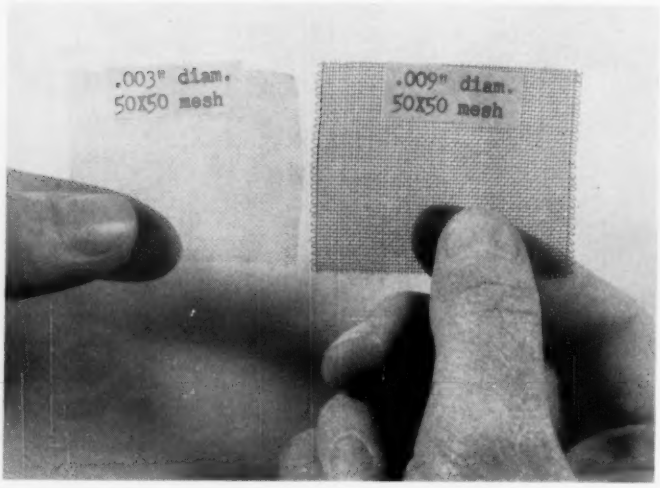

Fig. 11. — Photograph showing on the left a single thickness of the tantalum mesh gauze used in this study, and on the right a single thickness of the tantalum mesh gauze proposed for future use.

it is felt that the homograft contributes a useful service in that it forms a temporary lining and a wall which is not completely resolved for approximately one month, providing the necessary framework for proliferating fibrous connective tissue which replaces it and also infiltrates the tantalum mesh gauze. According to Jackson,[6] preserved tracheal homografts are better than fresh ones, for the preserved homografts are not viable and do not excite so severe a tissue reaction within the host as do fresh homografts. The limitation posed by the use of tantalum mesh alone instead of a homograft supported by tantalum mesh is illustrated by the results of a recent study which reported that with the use of tantalum mesh alone no dog survived longer than seven weeks.[11]

The relative success of the anastomoses of the tracheal autografts is interesting, for tracheal autografts have previously been reported to be unsatisfactory.[2] Of course, it would not be possible in man to repair tracheal defects with tracheal autografts. The ideal method of tracheal repair would be to dispense altogether with a graft and simply resect the desired length of trachea and do a primary end-to-end anastomosis. In man, this is possible only for resected lengths of trachea of not more than two to three tracheal rings (approximately 2 cm.).[20]

Infection is sure to destroy all efforts to obtain a satisfactory result. Cortisone has no value in aiding the host's tissues to accept the homograft. In one dog the cortisone seemed to have initiated infection of the graft.

The best method of tracheal anastomosis is with interrupted 34-gauge wire suture through the cartilaginous portion and with a continuous row of 000 absorbable surgical sutures through the membranous portion. With this method of anastomosis there have been no granulation tissue polyps about the suture lines.

Ciliated respiratory epithelium could not be demonstrated to line the homografts. The ciliae present in respiratory epithelium are necessary to propel the mucous blanket from the bronchial tree to the larynx. One would anticipate that because of the absence of ciliae a 3-4 cm.-long sleeve of stratified squamous epithelium or tall columnar epithelium would prevent this necessary toilet of the trachea.

Additional methods of tracheal grafting should be explored, using the above method of anastomosis. For example, the tantalum mesh might be lined by a split-thickness skin graft or a cylinder of nylon plastic.

Summary

A method is described for tracheal sleeve grafting in dogs, using tracheal homografts supported by tantalum mesh gauze.

Anastomosis is performed, using interrupted sutures of 34-gauge steel wire through the cartilaginous portion and a continuous row of 3-0 absorbable surgical sutures through the membranous portion.

The length of the homografts varied from three to seven tracheal rings (approximately 2 to 4 cm.).

The dogs in which the tracheal homografts were not wrapped with the tantalum mesh gauze for support averaged five weeks after grafting before they died of asphyxia because of stenosis and collapse of the trachea at the site of grafting.

The majority of the dogs in which the tracheal homografts were wrapped for support with tantalum mesh gauze averaged six months postoperatively before showing some stenosis of the three- to five-ring grafts (approximately 2-3 cm.) and three to four months postoperatively before showing stenosis of the seven-ring grafts (approximately 4 cm.). Presently there is a dog in the 13th postoperative month with a five-ring tracheal homograft which does not as yet show significant stenosis.

The stenosis of the trachea was due to contraction of the newly formed fibrous connective tissue, producing buckling of the supporting wrap of tantalum mesh gauze.

Cortisone did not retard the initial severe tissue reaction of the host to the viable homograft.

Repeated tracheal dilatations produced only temporary relief of the symptoms associated with the tracheal stenosis.

Two to four months were required for stratified squamous epithelium to replace the granulation tissue as the lining membrane of the graft. If the inflammation resolved, columnar epithelium would line the graft three to six months postoperatively. Respiratory-type epithelium was not found in the lining membrane of the grafts.

The homografts were wrapped with a double thickness of 0.003 in. diameter tantalum wire gauze of a 50×50 mesh. It is planned that future homografts will be nonviable and will be wrapped with a single layer of 0.009 in. tantalum wire gauze of 50×50 mesh, thus producing stronger support for the graft.

Dr. Ray Shirley and the Radiology Department assisted with the tracheograms. Mr. Dan Wiltfang assisted in the preparation and care of the animals.

University Hospitals, Dept. of Otolaryngology and Maxillofacial Surgery.

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Leiomyomata of the Esophagus

H. W. LUEDERS, M.D.; A. STRANAHAN, M.D.; R. D. ALLEY, M.D.; H. W. KAUSEL, M.D., and A. S. PECK, M.D., C.M., Albany, N. Y.

Of the several hundred leiomyomata of the esophagus reported, only eight cases have been discovered with an associated diverticulum. Five of the eight were originally described as autopsy findings,[13,1] while more recently three have been clinically evaluated and surgically removed.[11, 12,15] Of interest also is the frequency of an associated hiatus hernia with those leiomyomata occurring in the lower third of the esophagus. Eight such cases have been reported, and it is speculative what might be the causal relationship.

This paper will make no attempt to review the literature since an excellent detailed summary has been prepared by Lewis and Maxfield[1] but rather will report on four cases and their findings. Of particular interest is one case of an associated diverticulum and a calcified leiomyoma; two other cases are incidental findings on thoracotomy and one was an autopsy finding.

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A résumé of cases follows:

CASE 1.—A 56-year-old white man was admitted with dysphagia of six months' duration without weight loss. Dry foods such as bread tended to stick in the esophagus, and fluids were required to wash down the bolus. Shortly before admission swallowing was accompanied by a sensation of substernal pain. On examination he was a well-developed man without any significant physical findings. Roentgenograms of the chest, however, demonstrated a smooth and spherical soft tissue mass in the region of the esophagus at the junction of the upper and middle thirds, causing compression and posterior displacement of the esophageal lumen and a moderate delay in the passage of the barium beyond the tumor (Fig. 1). Bronchoscopy showed no tracheoesophageal fistula or other abnormalities. Esophagoscopy revealed a fungating lesion at 29 cm., which on biopsy contained considerable calcific material and histologically was read as an active chronic ulcer. Because of the suspicious nature of the lesion, the patient was reesophagoscoped, and the additional biopsies again

Fig. 1. (Case 2).—Close-up of the roentgenogram of the esophagus showing the leiomyoma with the diverticulum. Areas of calcification do not visualize well. The mucosal defect and mouth of the diverticulum are shown.

Fig. 2 (Case 2).—Cross section through the specimen shows the diverticulum filled with inflammatory and necrotic material. The squamous epithelium is eroded away. Calcifications are easily discernible within the leiomyoma.

revealed chronically inflamed fibrous tissue containing pigment and calcium without evidence of neoplastic cells. Since no definite diagnosis could be made from these studies, the patient was sent home to be followed as an outpatient. He returned five days later with nausea and anorexia, and one day had hematemasis productive of several clots and coffee-ground material. Physical examination was unchanged save for a slight pallor, a hemic apical systolic murmur, and melena on rectal examination. Fluoroscopy of the esophagus showed no change from the previous examination, so a preoperative diagnosis of benign tumor was entertained. On right exploratory thoracotomy a firm partially calcified tumor was found arising in the esophageal wall just below the azygos vein. When the esophagus was opened longitudinally the mucosa was found eroded, so it was necessary to partially excise a portion of the esophageal wall along with the tumor. Frozen section proved the lesion to be benign. The defect was repaired by transverse closure in layers, covered with pleura, and the chest closed. A Levin tube was introduced under direct vision into the stomach where it remained until the third postoperative day. The patient's subsequent course was uneventful, and on follow-up he was symptom-free, with no barium evidence of stenosis at the site of local excision. Pathological examination of the resected specimen revealed a calcified leiomyoma of the esophagus and formation of a traction diverticulum, which was ulcerated and contained calcareous masses (Fig. 2).

Fig. 3 (Case 1).—Roentgenogram showing the hiatus hernia.

Case 2.—A 68-year-old white woman, with a typical history of hiatus hernia, entered the hospital becasue of epigastric pain of eight months' duration, associated with anorexia and a 30 lb. weight loss. Physical examination was negative except for evident debilitation. Laboratory studies and pulmonary function tests were all within normal limits. Esophagram was not performed, but an upper gastrointestinal series revealed a hiatus hernia without demonstration of any tumor (Fig. 3). Through a left transthoracic approach the hiatus hernia was visualized and mobilized, at which time a small ring-like tumor of the distal third of the esophageal wall measuring 3×0.8×0.3 cm. was encountered and enucleated without entering the esophageal lumen. The esophageal muscle fibers were approximated, and the hernia

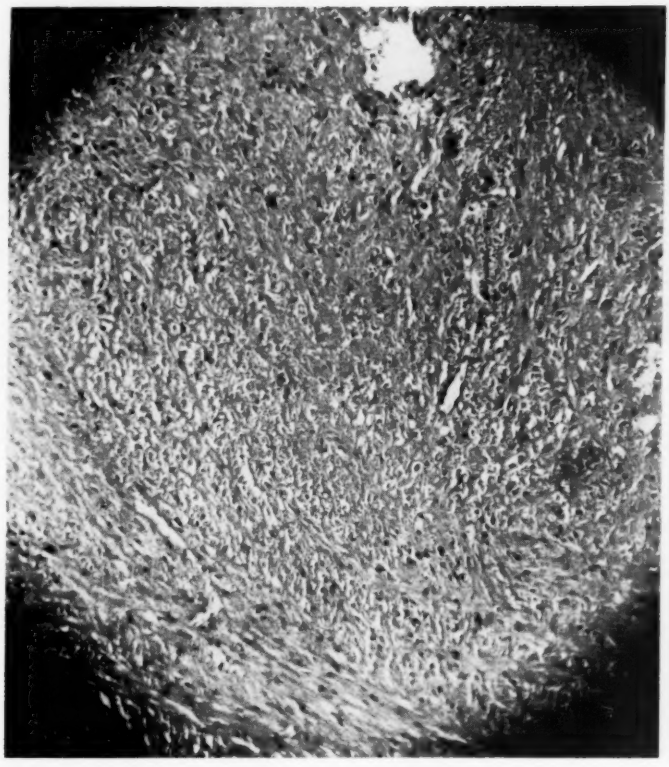

Fig. 4 (Case 1).—Photomicrograph of the leiomyoma. Hematoxylin and eosin; reduced about 12% from mag. × 40.

was then repaired. Since surgery in 1951, she has remained asymptomatic. Grossly, the tumor was yellow-white, firm, and pouting with tight whorls; microscopic examination revealed it to be a leiomyoma (Fig. 4).

CASE 3.—A 49-year-old white man was admitted with a history of sharp burning pain of the nailbeds of the ring and long fingers of two weeks' duration, unrelieved by narcotics. The right hand showed moderately severe vascular insufficiency, relieved to a degree by a sympathetic block. On performance of a cervicosympathectomy through the bed of the fifth rib, an incidental intramural tumor was found encompassing three-quarters of the circumference of the midesophagus. Frozen section and later permanent section revealed it to

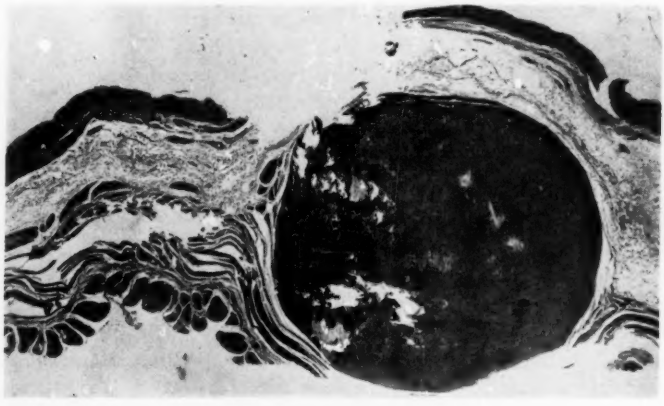

Fig. 5 (Case 4).—Photomicrograph of the small encapsulated leiomyoma within the wall, blending in with the circular fibers. Hematoxylin and eosin; reduced about 37% from mag. × 10.

be a leiomyoma. The tumor was dissected out, leaving the mucosa intact and closing the muscle primarily. The specimen measured 4×3.5×2.5 cm. and grossly showed a smooth pink to gray surface with abundant fibrous bundles. At no time did the patient have dysphagia or any of the other symptoms of esophageal compression.

CASE 4.—A 57-year-old white man was admitted for right-sided paralysis secondary to metastatic bronchogenic carcinoma to the brain. The primary lesion of the right lower lung had been resected two years previously. During his hospital stay the patient received supportive therapy. However, he died with an acute episode of bronchopneumonia. On postmortem examination, two nodules 1 and 0.4 cm., respectively, were found in the posterior wall of the esophagus just above the bifurcation of the trachea. Microscopic sections showed them to be leiomyomata (Fig. 5).

Comment

Leiomyomata are the most commonly reported benign esophageal tumors, varying from 0% to 0.4%, depending upon the particular autopsy series.[2-4] They occur as frequently in men as in women of any age group, and the average age of those patients treated surgically is around 40 years. Leiomyomata occur anywhere along the esophagus, either singularly or as multiple nodules, and may be either pedunculated or intramural. The pedunculated tumors are more frequent in the upper third of the esophagus and tend to ulcerate and bleed, and occasionally are discovered when they are regurgitated or coughed into the mouth. The intramural tumors occur more commonly in the middle and lower thirds of the esophagus, rarely ulcerate, bleed, or calcify, and sometimes are associated with diaphragmatic hernias. Usually they are asymptomatic, but they will produce symptoms late when they encircle the esophagus and obstruct it or the adjacent bronchi. Malignant degeneration has been observed, but this complication is rare.

The diagnosis may be suspected when symptoms of dysphagia, substernal or chest pain, regurgitation, vague epigastric distress, and anorexia appear and can be associated with fluoroscopic findings of mediastinal densities which may or may not move with deglutition. On barium swallow a horizontal shelf-like appearance at the junction of the normal and involved esophageal walls is considered to be diagnostic.[6] Esophagoscopy is frequently negative, showing only the smooth intact mucosa and extrinsic compression by tumor. Where the diagnosis is suspected, biopsy is not recommended, since it impairs the later enucleation of the tumor and may result in bleeding or infection. Bronchoscopy should be done in high lesions to rule out fistulous connection with a primary tumor in the tracheobronchial tree.

The associated findings of a diverticulum with a leiomyoma has no clear-cut origin. Stewart[13] mentions a common congenital origin of imbedded gastric mucosal rests which give rise to the diverticulum and the leiomyomatous growths. Mechanical factors appear to play the greatest part. The weight of some of the tumors alone as well as the repeated push of each bolus of food as it passes by could easily give rise to a pulsion-traction type of diverticulum. Whether a diverticulum occurs first and is later encompassed by the reactive growth of the surrounding muscles is debatable since in most cases the symptoms of a diverticulum are not obtained.

The incidental finding of a leiomyoma discovered when repairing a hiatus hernia is not frequent enough to be significant,[4-9] but it may help in explaining the pathogenesis. The tumor is in the lower third of the esophagus and might arise from the abnormal tensions placed upon it. Most of the leiomyomata have been seen to arise from the inner circular muscular coat. These muscle fibers have a limit of distensibility which produces a small rupture which is repaired with fibrous tissue. This process is repeated numerous times until the overgrowth of fibrous tissue of smooth muscle is continuous enough to form a tumor which perpetuates itself along the circular coat to eventually encircle the esophagus.

Treatment is usually effected by simple enucleation without disturbing the mucosa

and reapproximation of the muscular wall. If the tumor is so large as to encroach upon the lumen, partial resection or complete resection may be done. If malignant degeneration is suspected or proved, radical treatment is indicated. However, malignant degeneration is rare in the leiomyomata of the esophagus, particularly when compared with those arising in the stomach or small intestine.

Summary

Four cases of leiomyomata of the esophagus have been presented. One case is of special interest since the leiomyoma was calcified and had an associated diverticulum. The apparent frequency of hiatus hernia with leiomyomata is brought forth, and the etiology of both the diverticulum and the leiomyoma in hiatus hernia is submitted.

Leiomyomata of the esophagus are relatively rare but must be considered in the differential diagnosis of esophageal neoplasms. Therefore, a brief résumé of the pertinent diagnostic signs, symptoms, and procedures is given as well as the usual treatment.

Dr. E. A. Jacobs of the Columbia Memorial Hospital, Hudson, N. Y., gave permission for the use of the roentgenograms reproduced in this article. Dr. Pelham Glasier supplied information on Case 2, and Dr. John H. P. Holden on Case 3. Case 4 is taken from the files of the Veterans' Administration Hospital in Albany, N. Y.

Hun Pavilion, Albany Hospital (Dr. Stranahan).

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The Posterior Pharyngeal Flap Palatoplasty

G. OBREGON, M.D., Iowa City

The posterior pharyngeal flap palatoplasty is by no means new. Trendelenburg first conceived the idea and obtained successful results in animal experimentation.

Passavant [1] in 1865 was the first to raise a quadrilateral flap on the posterior pharyngeal wall, fold it on itself, and produce a pad. Later Garel [2] and Passavant advised the use of a palatal obturator. Schoenborn [3] and Schede [4] in the late 19th century advocated the pharyngeal flap operation, but it fell into disrepute because of numerous failures.

Gersuny [5] and Eckstein [6] proposed the injection of paraffin into the retropharyngeal space, while von Gaza [7] produced a pad in the posterior pharyngeal wall by transplantation of fat and fascia into the retropharyngeal space.

The pharyngeal flap was revived by Rosenthal [8] in 1924 and introduced in this country by Padgett [9-11] in 1930; at that time he reported a group of cases in which there had been considerable loss of palatal tissue as a result of previous surgery. In such cases the operation was followed by prompt speech improvement. Several authors [12-17] have written and contributed to a better understanding of this problem.

The pharyngeal flap operation is indicated whenever there is a defective palatopharyngeal closure. This is observed in 40% of repaired cleft palates, in paralysis of the soft palate, in congenital short palate, and in submucosal clefts.

The crux of the problem is to determine when a patient has defective velopharyngeal closure. This requires expert speech and orthodontic consultation. Lateral x-rays of the head and neck are needed to visualize the relation of the soft palate to the posterior pharyngeal wall during production of sounds such as "u," "s," etc. Pressure recordings are also required to evaluate the efficacy of velopharyngeal closure.

The problem is not one of "closing a hole" but one that requires the expert advice of otologist, surgeon, speech pathologist, orthodontist, psychologist, and social worker. Few medical problems call for such intimate cooperation of different specialists, if the patient is to receive maximum benefit from present-day knowledge.

One author [14] advises that the operation be postponed until the patient is 8 years old to allow time for physiological involution of the adenoids. In our clinic we do not believe this to be an important factor. If the operation is indicated, an adenoidectomy is performed three months prior to the pharyngeal flap operation. In special cases we use a turnover flap technique and leave the adenoids attached to the pharyngeal flap.

Some authors [12] believe the pharyngeal flap procedure alone is sufficient to attain satisfactory results while others [13,20] prefer a combination of the push-back procedure and a pharyngeal flap operation at the same time. We do not believe that any one type of operation is indicated for all cases but that the patient should be individualized and the operation planned for his particular needs.

A mucosal flap leaving the pharyngeal muscles undisturbed for fear of interference with the musculature of the pharynx and function of the Eustachian tubes has been advocated.[21] We incorporate the pharyngeal musculature in our flaps and have not observed any untoward effects.

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Assistant Professor, Department of Otolaryngology and Maxillofacial Surgery, State University of Iowa.

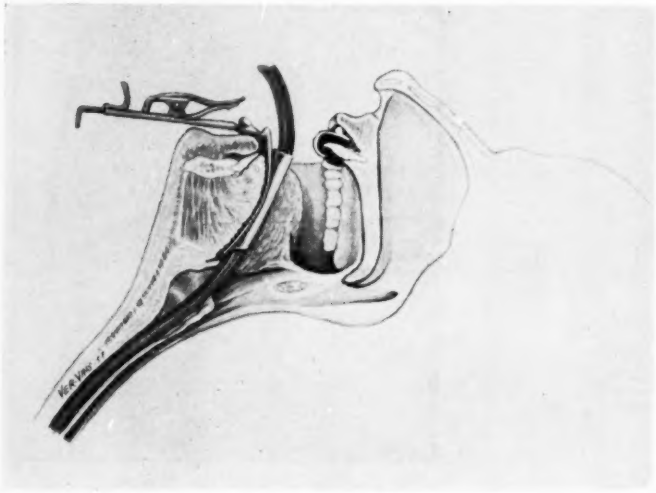


Fig. 1.—The excellent exposure provided by the Barton-Young mouth gag is well demonstrated in this drawing.

Pharyngeal flaps can be based nasally or orally. The palatal bed can be prepared by denuding, splitting, grooving the edge of the soft palate, or by using a turn-over palatal flap.

In our clinic we formerly denuded the edge of the soft palate and sutured the pharyngeal flap to the prepared palatal bed. However, we were not satisfied with our immediate postoperative results.

After we became acquainted with the imbricated flap [18] it occurred to us that the same principle could be used in the pharyngeal flap operation. A review of the literature on this subject revealed an article [19] in which the method that we have been using was mentioned. However, no discussion of emphasis was placed on the advantages of this technique.

Technique of Posterior Pharyngeal Flap Palatoplasty

1. The patient is given a nonexplosive anesthetic agent by way of an endotracheal tube. This agent enables us to use electrocoagulation.

2. The head of the patient is hyperextended and a Barton-Young mouth gag is used. It provides

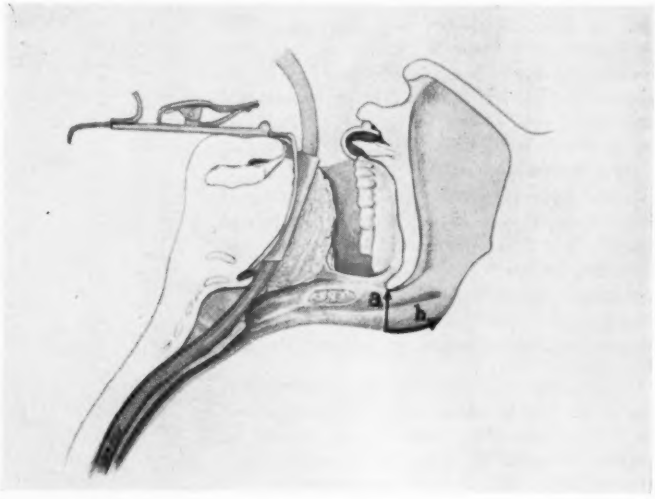


Fig. 2.—The arrow *a* indicates the velopharyngeal distance; *b* shows the incorporation of this distance to the cephalic portion of the pharyngeal flap.

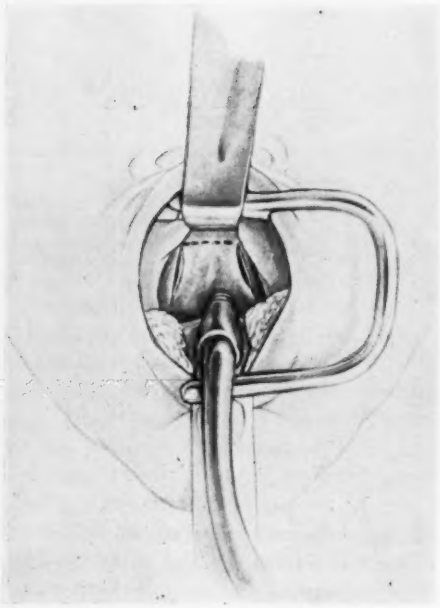


Fig. 3.—The nasopharynx is exposed with a palate retractor. The lateral incisions of the pharyngeal flap are completed. The dotted line indicates the superior incision of the pharyngeal flap.

excellent exposure by retracting the tongue and keeping the endotracheal tube out of the operative field* (Fig. 1).

3. We prefer to base our pharyngeal flaps orally because better speech is obtained as a result of lowering the soft palate to the position where most of the stop plosive sounds are formed. The surgeon sits at the head of the patient, and a headlight is used throughout the operation. The velopharyngeal distance is determined, and this measurement should be employed whenever possible in calculating the extension of the pharyngeal flap above the posterior edge of the soft palate (Fig. 2). If this is not feasible, the pedicle of the flap is left attached superiorly.

4. The posterior pharyngeal wall is infiltrated with a small amount of procaine and epinephrine solution. The infiltration facilitates dissection and minimizes bleeding.

5. With a No. 11 Bard-Parker blade mounted in a long knife handle two parallel incisions located as far laterally as possible are made in the posterior pharyngeal wall (Fig. 3). Mucosa, pharyngeal muscle, and aponeurosis are included.

6. The flap is elevated from the pervertebral fascia with a dissector. Small bleeding points are electrocoagulated.

7. The soft palate is retracted, and the cephalic attachment of the pharyngeal flap is severed with scissors or knife.

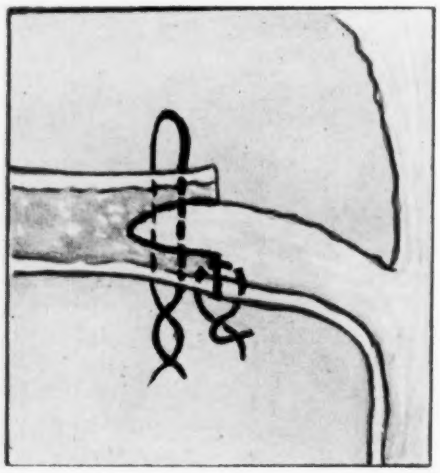
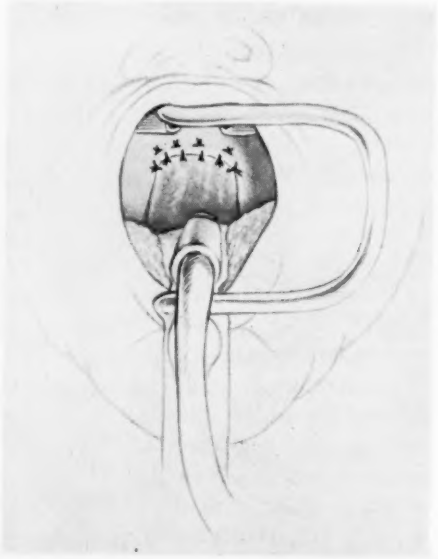


Fig. 4.—Side view of the pharyngeal flap attached to the soft palate, using the imbricated flap technique. Through-and-through mattress sutures and edge-to-edge approximation.

Fig. 5.—Completion of the operation with pharyngeal flap anchored in place.



* One author [14] has advocated the use of tracheotomy at the time of operation but in our experience we find it unnecessary; others believe that the operation is extremely difficult under general anesthesia. This has not been our experience.

8. From 1 to 1.5 cm. of oral mucosa of the cephalic portion of the pharyngeal flap is removed (Fig. 4).

9. The soft palate is split so that the oral and nasal mucosae can be separated to receive the denuded pharyngeal flap.

10. The flap is anchored in place without tension with mattress sutures. Another row of interrupted sutures is placed to coapt the mucosal edges of the palate and pharyngeal flap. (Fig. 5).

Results of a series of posterior pharyngeal flap palatoplasties will be published in a forthcoming article.

Summary

A brief review of the history of the posterior pharyngeal flap palatoplasty is presented.

The different techniques used in the pharyngeal flap operation are mentioned.

The use of the imbricated flap technique is advocated in the pharyngeal flap operation.

The technique used in our clinic for the posterior pharyngeal flap palatoplasty is described in detail.

University Hospital.

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Pharyngeal Diverticula

W. R. DENNY, F.R.C.S., and C. P. WILSON, F.R.C.S., London

Innumerable articles have been written on the subject of pharyngeal diverticula since Ludlow, in 1764, first drew attention to this condition.

It is not the object of this paper to discuss features to which attention has been drawn on many occasions, but to analyze, firstly, the suggestions which have been advanced as being factors in the production of pharyngeal pouches, and, secondly, to suggest a technique of operation which in our view not only makes operation far simpler but also makes the end-result more certain and permanent.

Anatomically, there are two potentially weak areas in the posterior pharyngeal wall, one below and the other above the cricopharyngeus muscle.

The lower area is caused by the forward convergence of the longitudinal muscle of the esophagus to gain anterior attachment to the middle line of the posterior surface of the cricoid cartilage. The triangular area formed by this deviation of the longitudinal musculature is known as the Lannier-Hackerman point. This area is of theoretical weakness only as the circular fibers of the gullet are well able to resist such increased esophageal pressure as may occur. The area is not exposed to increased pharyngeal pressure because of its protection superiorly by the cricopharyngeal sphincter. Above the cricopharyngeus there is another triangular area of potential weakness formed by the tendency to divergence of the oblique fibers of the inferior constrictor from the transverse fibers which form the cricopharyngeal muscle. This area is usually named after Killian (1908), who described it in 1908, although it was previously described by Ludlow in 1764.

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In the normal dissected specimen one cannot demonstrate any actual triangular area devoid of muscle fibers, but for some distance above the transverse fibers of the cricopharyngeus muscles the fibers of the inferior constrictor become more and more oblique so that the area is less well buttressed against pressure than in areas where the fibers are parallel. It is from this area that the common type of pharyngeal diverticula arise.

There is, unfortunately, much confusion about the nomenclature of these weak areas, and even Mosher in 1917 describes the lower weak area—the Lannier-Hackerman point—as Killian's area.

Although few dispute the site of origin of pharyngeal diverticula, a study of the literature shows that similar unanimity does not apply to either the method of production or the treatment, and it is to these two aspects of the condition that we intend to confine our remarks.

Etiology

In discussing the etiology of pharyngeal diverticula the main concern of most writers has been the exact site of origin rather than the mode of origin. The site has now without any doubt been firmly established as the upper area, Killian's (or Ludlow's) area, and discussion centers mainly on the mode of production of the pouch.

It has been held that pharyngeal diverticula have a congenital origin, and arise from a congenital dilatation of the pharynx, with relative weakness between fibers of the inferior constrictor muscle and the cricopharyngeus, and that repeated forceful swallows cause at first bulging and ultimately sac formation. This theory received backing from Dunhill (1950), who reported one

family among whose members six developed pharyngeal pouches.

Hudson (1951) proposed another congenital theory. He suggested that pharyngeal pouches were, in fact, true congenital diverticula. He reached his conclusion because in the normal hypopharynx and upper esophagus he found it was impossible to distinguish individual musculature or to demonstrate areas of potential weakness. King (1947) denied the congenital origin. All the cases of which he knew arose only after mature development of the larynx. He believed that pressure between the fifth cervical vertebral point and the lower border of the cricoid cartilage led to thinning or degeneration of the muscular fibers of the inferior constrictor muscle forming the posterior pharyngeal wall. It was this thin area which allowed herniation.

Negus (1950) associated the origin of pharyngeal pouches with human evolution. The assumption of the erect posture in man straightened the cervical spine and caused recession of the mandible. This in turn led to descent of the larynx. As a result the upper part of the inferior constrictor muscle became stretched and oblique, thus leading to potential weakness in its lower and posterior part. Owing to the descent of the epiglottis, inspired air could reach the larynx and hypopharynx as easily through the mouth as through the nose, and because of the large size of the hypopharynx as compared with the larynx a sphincter had to be developed to prevent air entering the esophagus on respiration. Strong arm movements required the closure of the glottis, and the sphincter at the upper end of the esophagus was also needed to prevent entry of air into the esophagus during such exertions. Man had, therefore, a cricopharyngeal sphincter and also a weakened area in the posterior part of the hypopharynx. Negus' explanation of the production of a pouch was that during the second stage of the swallowing act the cricopharyngeus failed to relax; as a result there was a sharp rise in intrapharyngeal pressure and eventually a herniation of the pharyngeal mucosa. No entirely satisfactory explanation is given as to why the cricopharyngeus should fail to relax.

It is probable that several factors may be involved in the etiology, and if two or more of these factors are present a pouch may result.

There may be a congenital influence. For instance, a congenitally large lax pharynx with thinly spread muscle will in itself be naturally associated with a weakness of Killian's area. A true congenital diverticulum is described, but it has a complete and well-developed muscle coat, and it is unlikely that such a congenital pouch is a frequent antecedent of the pharyngeal diverticulum commonly seen, which has a very sparse and thinly disposed muscular coat.

Increased pressure in the pharynx must undoubtedly be a major factor in the initiation of a diverticulum by bulging the weak part of the posterior wall, and the problem would appear to be one of deciding why and how such increased pressure may be produced.

Once a pouch exists, no doubt the actual weight of its contents will tend to cause progressive enlargement, but this naturally has no bearing on the initial production of the pouch.

Pharyngeal pouches are commoner in men than in women; men have a larger cricoid cartilage, and a large bulging posterior surface of the cricoid cartilage will presumably tend to divert the food bolus on to the posterior wall of the hypopharynx.

Cervical disk degeneration is a common condition. It occurs most frequently between the fifth and sixth cervical vertebrae and is usually associated with osteophyte formation. This may lead to indentation of the posterior wall of the pharynx and possibly spasm of the cricopharyngeus muscle. However, all men do not get pouches although they may have large cricoid cartilages, nor do all persons with cervical disk degeneration get pouches, although it is rare to find a patient with a pouch who does not

exhibit such a condition. These factors may be ancillary but cannot be accepted as being anything more than incidental predisposing factors in the production of the pouch. One must accept that the main factor is an increased pressure in the hypopharynx and this can occur only if there is an associated closure of the cricopharyngeal sphincter.

It is not good enough to postulate that in these cases there is an idiopathic neuromuscular dysfunction associated with cricopharyngeal spasm unless one can give a reasonable explanation of why this dysfunction should occur. We are of the opinion that the solution of the problem is to be found in a study of the physiology of swallowing.

In the normal swallowing of a bolus of food there are, as is well known, three separate phases. Firstly, the placing of the bolus on the back of the tongue to initiate the act of swallowing. Secondly, the shooting of the bolus of food—not into the pharynx—but directly into the gullet. This is made possible by the relaxation of the inferior constrictor and cricopharyngeus muscles, associated with enlargement of the pharyngeal lumen. This enlargement is aided by the elevation of the larynx, which makes the fibers of the constrictor more horizontal. Whether this shooting of the bolus of food is due to simple muscular action of the tongue or associated with an airgun action of a bubble of air trapped by the tongue, as suggested by Podvinec (1952) is immaterial. The essential feature is that the passage of the bolus through the pharynx is of great rapidity and is facilitated by the relaxation and enlargement of the hypopharynx. Once the food bolus reaches the gullet, the cricopharyngeus goes into active contraction and remains in active contraction while there is anything in the upper gullet. When the upper gullet is empty, it returns to its normal condition of tonic contraction. Now if there should be a second act of swallowing, or a contraction of the pharyngeal muscles during this period of active spasmodic contraction of the cricopharyngeal sphincter, then the condition is established of increased pressure in the hypopharynx, which is the primary condition postulated as being favorable to the development of a pouch or hernia.

What conditions may produce this effect? We suggest, firstly, rapid and repeated acts of swallowing, such as are produced by swallowing a glass of water or other fluid very quickly; the second or third or fourth gulps will certainly occur at a time when there is still something in the upper gullet and the cricopharyngeus is actively contracted. It is a procedure that without any question is more frequently indulged in by men than women. The second factor we suggest as a possibility is a curious one and one to which, as far as we are aware, attention has not previously been drawn.

If one makes a conscious effort of swallowing one's saliva, there is no bolus; the salivary blob is shot into the pharynx and is likely to stick to the posterior wall of the hypopharynx rather than to be projected into the gullet. At the same time the associated air bubble will most probably pass into the gullet. The result will be that there will be a bubble of air in the upper gullet resulting in active cricopharyngeal contraction while at the same time there will be a blob of salivary mucus in the posterior pharyngeal wall. In four out of five normal persons whom we have asked to make a conscious action of swallowing saliva, we have found that the intitial act of swallowing is almost immediately succeeded by a subsidiary act of swallowing. This subsidiary swallow is of an entirely different nature from that of the initial swallow and is simply produced by contraction of the inferior constrictor and pharyngeal muscles. Anyone can demonstrate this phenomenon for himself quite easily by swallowing his saliva consciously, and it will be generally found that the primary swallow is succeeded by a subsidiary swallow which feels quite different from the primary one. If two or three such swallows are made in rapid succession, the cricopharyngeal spasm be-

comes sufficiently marked to make one conscious of a lump in the throat—the acute cricopharyngeal spasm associated with a bolus of air in the upper gullet.

Here, then, we have the ideal predisposing conditions—an acute spasmodic contraction of the cricopharyngeus with an accompanying active contraction of the inferior constrictor muscles, thus producing an increased air pressure in the hypopharynx.

We suggest, therefore, that the production of a pouch is possibly associated with the presence of one or more of the following factors:

1. A congenitally lax pharynx
2. A large cricoid cartilage
3. The habit of swallowing fluid in rapid succession
4. Eating too quickly
5. Cervical disk degeneration with arthritis of the cervical spine
6. A throat consciousness producing swallowing of air and saliva, which may be associated with Factor 5

The combination of two or more of these factors may produce the essential predisposing conditions of active contraction of the inferior constrictor muscle in the presence of spasm of the cricopharyngeal muscle with consequent increase in hypopharyngeal pressure. The type of swallowing which leaves some residue on the hypopharyngeal wall and which we might call an unclean swallow can be produced by a throat consciousness—much commoner in women—or by swallowing a bolus which has been inadequately prepared for deglutition by admixture with saliva, in other words, eating too quickly—much commoner in men.

Treatment

Assuming that the patient is a reasonably fit subject, the only satisfactory treatment must be of a surgical nature. Interference may have as its object palliation of the symptoms without removal of the pouch or removal of the pouch and repair of the pharyngeal wall.

Other methods of treatment have been used in the past, and Halsted, writing in 1904, mentioned some of these. He cites the injection of silver nitrate into the pouch—this case, not surprisingly, ended fatally. He mentions the passage of sounds and stomach tubes, the application of a faradic current—this led to a shortening of the sac by 3 cm. in four years—and gastrostomy.

Of the more rational forms of palliative treatment there is the now obsolete operation of diverticulopexy. As recently as 1950, Dunhill suggested that it might still be employed in a frail, elderly patient. The disadvantages are the marked tendency to recurrence of symptoms and the occasional development of carcinoma in the sac.

Invagination of the sac into the esophagus with suture of the muscle coats to repair the defect was described by Giraud in 1896. Bevan (1917) suggested obliteration of the sac with sutures and its subsequent invagination into the esophagus. A further modification of the invagination technique was proposed by Seiffert (1953). This consisted of the division of the cricopharyngeus with inversion of the sac by purse-string sutures. Invagination, like diverticulopexy, is often associated with recurrence of the pouch.

Mosher, in 1917, reported four successful cases of endoscopic division of the party wall, using a scissor punch. These were really astonishing results, considering that endoscopy was relatively new; the operation was carried out under local anesthesia; there were no antibiotics, and the wall was divided to within ⅛ in. of the bottom of the sac.

Dohlman (1949) similarly argued that if a diverticulum was caused by contraction of the cricopharyngeus or inability to open the sphincter, the treatment of choice was to cut the sphincter and enlarge the mouth of the diverticulum so that food would not accumulate in it. He obtained this objective by endoscopic division of the party wall between the pouch and esophagus by diathermy. Dohlman, Larsen (1953) and Spencer-Harrison (1956) have all reported successful results by this method and have not found mediastinitis a hazard.

For this technique it is claimed that it is simpler than external operation, involves

much less risk, requires a shorter stay in hospital, the symptoms are completely relieved, and recurrence does not take place. It is not a method that appeals to us and we would quote Mosher again— "The method of dissecting out the esophageal pouch is a thoroughly scientific procedure, perhaps the most scientific of all."

In the early days of operative removal of the pouch, the most serious complication was sepsis, with leaking from the breaking-down suture line leading to cervical or even mediastinal infection. This occurred in the original one-stage operation, and as a result a two-stage operation was devised. This varied in detail in the hands of different surgeons but basically allowed the sealing off of tissue planes before the sac was removed. This sealing off was often more theoretical than real, and when the second stage was carried out the hoped-for barriers against infection were not always found. At the present time, as a result of the meticulous preoperative cleansing of the pouch and the development of antibiotics, the fear of septic complications has, to a large extent, been overcome, and there has been a general return to the one-stage operation. A notable exception was Lahey (1953), who had very large experience of the surgical treatment of pharyngeal diverticula but remained a staunch adherent of the two-stage operation.

The incision employed by most surgeons is one along the anterior border of the sternomastoid extending from the level of the middle of the thyroid cartilage to the clavicle, although in recent years some surgeons have advocated a transverse or collar incision at the level of the lower border of the cricoid cartilage. The omohyoid muscle is completely divided and the sternothyroid partly divided. The middle thyroid vein is ligatured, and the lateral lobe of the thyroid gland is retracted forward. The inferior thyroid artery is identified and divided. The sac is found lying deeply behind the upper end of the gullet between the lateral lobe of the thyroid gland anteriorly and the carotid sheath laterally.

The sac is identified; the fascia covering it is incised, and the sac freed from its investments by traction and blunt dissection. When the sac is thus delivered, such muscular covering as it may possess is stripped back as far as the pharynx. It will possess a well-defined border below and in front guarded by the cricopharyngeus muscle, but the posterior surface will imperceptibly blend with the posterior pharyngeal wall. It therefore cannot often be said to have a real neck, although by pulling the sac horizontally an artificial neck will be formed at the lower edge of the inferior constrictor muscles. In outline the neck of the sac is piriform, the stalk or point being in the middle line of the posterior pharyngeal wall at a higher level than the blunt anterior or lower border. Up to this point the operation is comparatively easy in the majority of medium-sized pouches, although the difficulty may be increased with large mediastinal pouches or pouches of long standing with peridiverticular inflammation. It is also of greater difficulty in patients with short, thick necks.

The major difficulty now commences. The lateral lobe of the thyroid is retracted forward with the trachea and gullet, the surgeon alternating between encouraging his assistant to retract a little harder and warning him to be careful not to use too much pressure because of the danger of causing injury to the recurrent laryngeal nerve.

The suturing of the neck is done with difficulty because of the limited exposure of the posterior wall of the pharynx, and accurate repair of the muscular posterior wall is not always attained. A portion of the pouch may be left behind because the surgeon is afraid of narrowing the pharynx if he takes too much mucous membrane away.

Such a picture may be unfair when one considers a patient with a long, thin neck, but it is unfortunately only too common in those with thick, short necks. It was because of these difficult patients that a search was made for some improvement in tech-

nique. When a surgeon operates upon an inguinal hernia, he makes his incision over the inguinal canal and not over the scrotum. He desires to obtain the best possible access to the neck of the hernial sac and is not worried about the separation of the fundus. The same principle applies to a pharyngeal pouch. The approach should be to the neck of the sac rather than to the fundus of the sac. To suture the neck of the sac easily the operator must have good vision of the posterior pharyngeal wall. This can be obtained only by rotation of the cricoid cartilage. This is not provided by retraction of the lateral lobe of the thyroid gland with the trachea and gullet. Furthermore, to pull on the trachea and gullet is to pull the recurrent laryngeal nerve from a comparatively fixed point in the cricoid cartilage, which is dangerous.

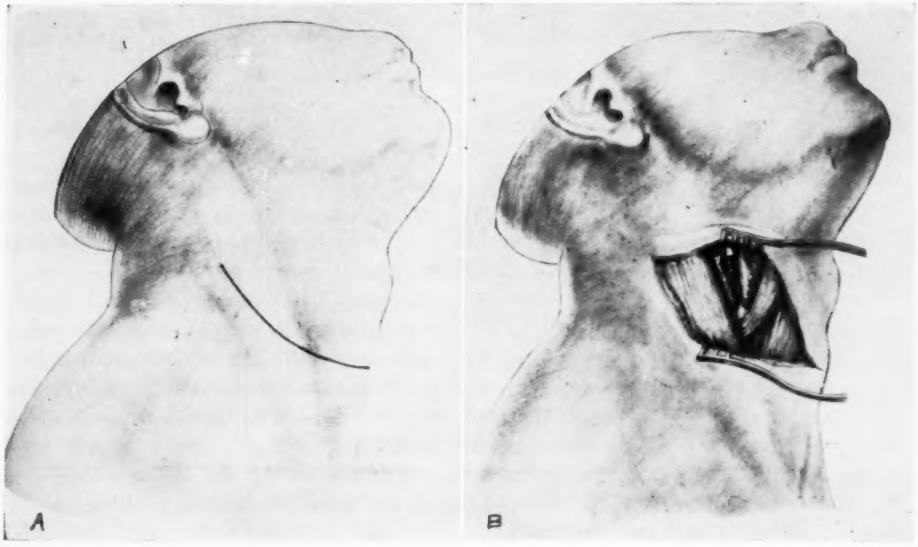
To avoid these difficulties, the cricoid cartilage must be rotated and this can only be done efficiently by rotating the thyroid cartilage. Not only does this give really good access to the posterior pharyngeal wall, but it does not endanger the recurrent laryngeal nerve, for rotating the cricoid pulls the nerve from mobile tissue and not from comparatively fixed tissues. As may easily be seen during a laryngectomy, the whole larynx can be rotated through more than 90 degrees by rotating the thyroid cartilage.

It has been suggested for many years that the sac should be approached from the left because it usually passes toward the left side. This is a myth which no one seems willing to dispel. In any case the neck of the sack commences in the middle line, and whether the fundus tends to deviate toward one side or the other is of no significance. We prefer to operate from the right side, as this is our normal position for laryngeal and oral operations. It is a matter of personal preference, access to the sac being equally good from either side.

The incision is a collar one, in a skin crease commencing laterally over the sternomastoid at the level of the upper border of the thyroid cartilage and curving downward and forward to reach the middle line at the lower border of the cricoid cartilage (Plate 1, *A*). The platysma is divided and the wound opened with a self-retaining retractor (Plate 1, *B*). The omohyoid muscle is di-

Plate 1

A, the collar incision.
B, after division of platysma the wound is opened with self-retaining retractors.



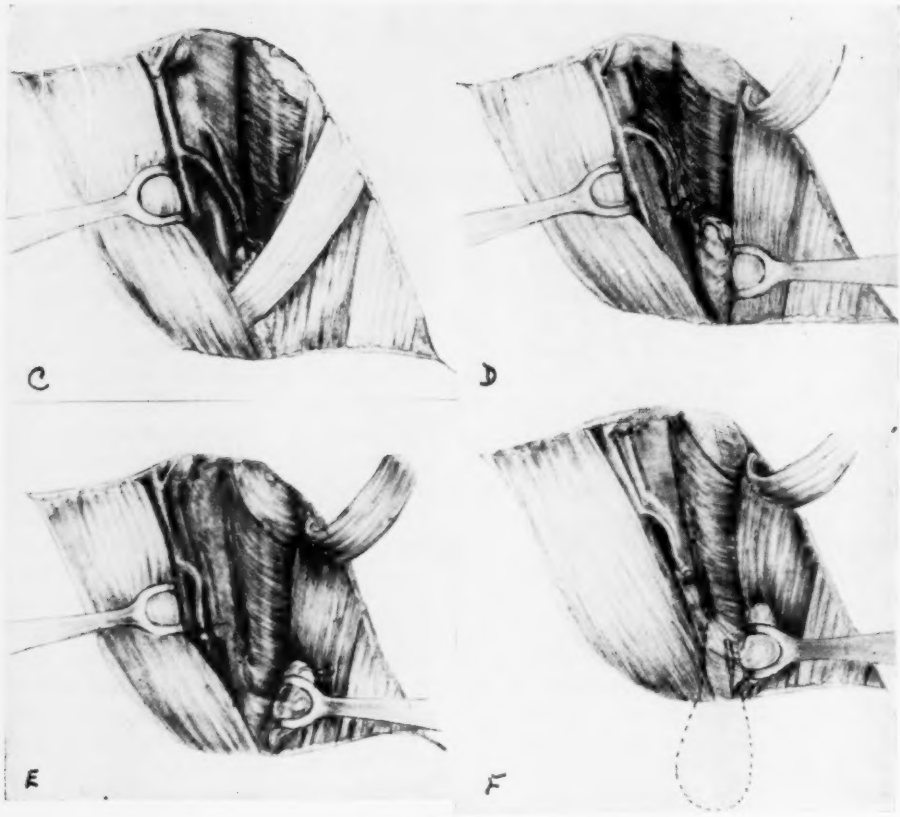


Plate 2

C, superior thyroid vessels and upper pole of lateral lobe of thyroid gland.
E, upper pole of lateral lobe of thyroid is freed and displaced forward and downward.
D, anterior belly of omohyoid is divided, and the superior thyroid vessels are ligated and divided.
F, the pharyngeal fascia covering the sac is divided.

vided at its tendon and the anterior belly dissected forward. The lateral border of the sternothyroid is identified and retracted foward; it need not be divided, although the outer edge can be divided high up if desired. The upper pole of the thyroid gland is seen, and the superior thyroid vessels or their branches are divided and tied (Plate 2, *C* and *D*). It is convenient also to divide and tie the middle thyroid vein.

The upper pole of the lateral lobe of the thyroid gland is freed from above and displaced forward and downward (Plate 2, *E*). The ala of the thyroid cartilage can now be felt throughout its extent, covered in its posterior part by the inferior constrictor muscle. By tracing the surface of the muscle downward one comes directly on to the lateral margin of the sac. At the beginning of the operation the pouch is packed through an esophageal speculum with ribbon gauze soaked in 1:1000 acriflavine solution. This causes the sac to bulge, and the color can be seen sufficiently well to distinguish it from the inferior constrictor muscle. The pharyngeal fascia covering the sac is divided with scissors (Plate 2, *F*); part of the packing is withdrawn by the anesthetist. This withdrawal makes identification of the sac certain. It is picked up with nontoothed

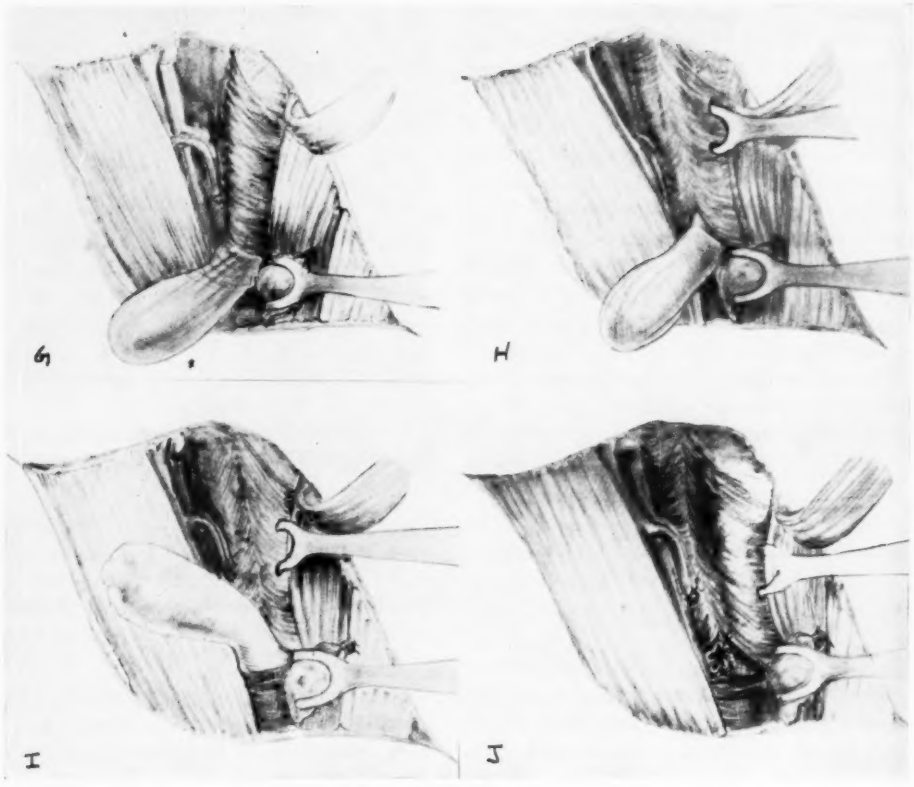


Plate 3

G, the sac has been freed from its fascial sheath.
I, cricopharyngeus is identified forming lower border of neck of sac.
H, muscle fibers covering outer part of sac are separated until neck is reached. The thyroid cartilage is rotated through 90 degrees to show posterior aspect of inferior constrictors.
J, the sac has been removed.

dissecting forceps in its sagging upper part and gently separated from within its fascial sheath by blunt dissection. The remainder of the gauze is pulled out and the sac freed (Plate 3, *G*). A blunt double hook is now placed behind the posterior border of the thyroid cartilage, and the thyroid cartilage is rotated through 90 degrees so as to present the posterior aspect of the inferior constrictor muscles to the operator. The pharyngeal fascia has already been divided over the sac, and this division is now continued upward so as to expose more clearly the actual muscle fibers of the inferior constrictors. It is this rotation of the thyroid cartilage that is the essential feature of the operation.

The muscle fibers covering the upper part of the sac are continuous with the lower fibers of the constrictor and are separated from the sac by blunt dissection until the neck is reached (Plate 3, *H*). The cricopharyngeus muscle is identified forming the lower border of the neck of the sac (Plate 3, *I*).

This part of the operation presents no difficulty because of the rotation of the thyroid cartilage. The recurrent nerve does not need to be identified; it is in no danger, being separated from the sac by the cricopharyngeus and below this by the whole thickness of the gullet; it is neither compressed by retractors nor pulled on.

It might be suggested that the incision recommended is inadequate in the case of a very large pouch, with adhesions due to repeated attacks of inflammation. This is a valid criticism, but the same difficulty will occur even with a vertical incision reaching the sternoclavicular joint if the pouch is sufficiently large for the fundus to be in the mediastinum. In such an event, there is no reason why an incision should not be made downward along the sternomastoid muscle from the original incision, and this we were obliged to do in one patient. This hazard, however, is concerned only with the freeing of a large adherent pouch and does not invalidate the importance of adequate access to the posterior part of the inferior constrictor muscles.

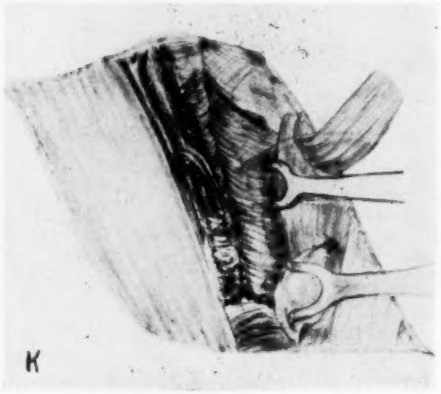


Plate 4

K, repair of inferior constrictors and obliteration of weak posterior triangle by inclusion of cricopharyngeus in the lower sutures.

Many variations in the technique of the actual excision of the sac have been practiced. Most of these are designed to prevent excessive narrowing of the reconstructed pharynx during the repair. We think this fear is much overrated. It would be very difficult to produce a stenosis of the pharynx, and in most patients with pharyngeal diverticula the pharynx is in any case much wider than normal. Because of this, we do not advocate the insertion of a bougie into the gullet.

Our own procedure for excision of the sac is to tie the neck of the sac with hardened absorbable surgical suture, making two turns round the sac and knotting between the turns. The sac is then transfixed distal to this ligature and tied again. The sac is removed distal to the transfixation ligature (Plate 3, *J*). The lower borders of the inferior constrictor muscles are now carefully sutured together. The lowest stitch includes the upper fibers of the cricopharyngeus, thus obliterating the weak posterior triangle (Plate 4, *K*). A few sutures are put into the pharyngeal fascia; the area of operation is sprayed with penicillin and sulfathiazole powder, and a small soft rubber drain is put into the incision about its middle. The skin wound is then closed round it. An antibiotic is given systemically for the first week, and the wound heals by first intention. A nasal feeding tube is not used postoperatively as it does not stop the patient from having to swallow his saliva and, in fact, it makes him want to do so more often.

Soft food can be given immediately after operation, but each meal should be completed by a drink. A soft diet is continued for 10 to 12 days, after which a barium swallow is performed. The patient is then allowed solid food and is able to go home at the end of two weeks.

We do not feel it is necessary to divide the cricopharyngeus and believe that such a division leads to the immediate production of a new weak point. As the muscle heals with a fibrous tissue junction, this is probably of no great moment.

We prefer tying off the sac as opposed to excision and suture because of the likelihood of perforating the pharyngeal mucosa by one of the stitches. If this occurs, the stitch hole may easily tear as soon as the stitch is tied, and as the mucous membrane in this situation is very thin and delicate the tear may become a centimeter or more long within seconds, and suture of this tear may cause not a little difficulty. No doubt every surgeon will have his own preference as to the method he uses, and we suggest the

method described as one which we have found most satisfactory in practice.

Summary

The possible factors in the production of pharyngeal diverticula are discussed, and it is suggested that a combination of two or more of these factors is likely to be operative if a diverticulum is to result. The difficulties of operation are outlined, and a technique is described which overcomes most of the difficulties which may be encountered.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

A Clinical Evaluation of Monaural and Binaural Hearing Aids

DONALD M. MARKLE, M.Ed., and WILLIAM ABER, M.A., New York

Introduction

At the Hearing and Speech Center of New York University-Bellevue Medical Center, a clinical evaluation of monaural and binaural hearing aids has been conducted.

Although much has been reported in the area of binaural vs. monaural hearing, the majority of previous investigators were concerned with either binaural summation of loudness or binaural summation at threshold. The development of miniature amplifiers which can easily be worn on the head, particularly the more recently developed "binaural hearing aid glasses," has stimulated a new interest in the advantages of binaural hearing.

Subjects and Procedure

Because of the many variables found in the nature of perceptive deafness (e. g., discrimination, recruitment, and tolerance), this investigation has been restricted to subjects with clinical otosclerosis; the hearing of 10 subjects has been investigated. These subjects produce audiograms which are consistent with the medical diagnosis and demonstrate no evidence of a discrimination problem. In effect, these subjects represent what is considered as being "ideal hearing aid candidates."

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The subjects were seated in the center of the test room of a conventional two-room suite and, 4 ft. away, and at a 45 degree angle to the left, was placed a loudspeaker (S) which is capable of delivering a signal, noise, or signal and noise, at calibrated levels. Four feet away, and at a 45 degree angle to the right, was placed a second loudspeaker (N) capable of producing recorded noise at calibrated levels (Fig. 1). Loudspeaker S is fed by an Allison 20-A hearing evaluation unit; loudspeaker N is fed by a Grason-Stadler 260-B auditory training unit.

In order to evaluate the relative values of monaural and binaural hearing, each subject was tested while wearing a conventional hearing aid in a conventional manner (Fig. 2). In addition, each subject was tested

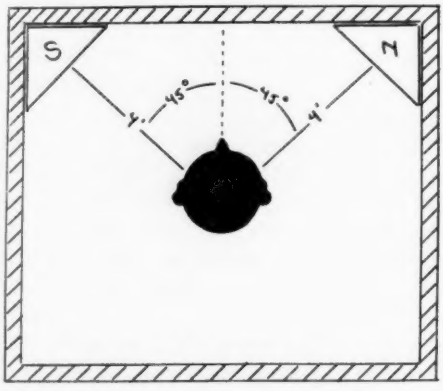

Fig. 1.—Diagram of test situation illustrating position of subject in relation to signal and noise transducers.

Fig. 2.—Subject wearing conventional hearing aid in conventional manner.

while wearing two conventional hearing aids —one mounted on each side of the head (Fig. 3). In each case, the subject was instructed to simply set the volume of the instruments to a comfortable listening level for 50 db. of connected discourse.

The test material consisted of spondee words (Auditory Test W-1) and phonetically balanced word lists (Auditory Test W-22), which were presented to the subject via "microphone voice" and which were carefully monitored by the examiner.

The noise used was recorded factory background noise (Standard Sound Effect Record 300-A), which is produced by Standard Radio Transcription Service, Inc.; this recording produces a constant acoustic output within ±1 db.

During the entire testing procedure, the noise was kept at a constant 60 db. sound pressure level; this level was determined by measurements made with a General Radio 759-B sound-level meter.* In the presence of this noise, speech reception thresholds were obtained; in addition, phonetically balanced word lists were presented at levels of

Fig. 3.—Subject wearing two conventional hearing aids—one mounted on each side of the head.

10 db. above the noise, at the same intensity as the noise, and at 10 db. below the noise; this represents signal-noise ratios of +10, 0, and −10. The subjects' discrimination scores were obtained at each of these levels.

Results

No significant difference was demonstrated between speech reception thresholds obtained with the use of monaural and binaural hearing aids (Table 1). At a signal-

TABLE 1.—*Comparative Scores of Monaural and Binaural Hearing Aids with Signal and Noise Coming from Different Directions*

	Monaural		Binaural	
	Mean	Range	Mean	Range
Speech reception threshold	43.8 db.	39–47	42.0 db.	39–45
Discrimination in noise				
S/N +10	88.0%	84–92	92.4%	90–94
S/N 0	66.4%	58–74	77.6%	76–80
S/N −10	4.0%	0–14	33.8%	28–44

* "C" weighting network.

noise ratio of +10, the results suggest a slight difference in discrimination with binaural hearing being favored. At signal-noise ratios of 0 and −10, however, the differences are significant and apparent.

Clinical Implications

In conventional hearing aid evaluation procedures, the evaluation of the performance of a hearing aid in the presence of noise is usually done while both signal and noise are presented through a common loudspeaker. In an effort to evaluate the use of conventional clinical instrumentation as a means of demonstrating the advantages of binaural over monaural hearing aids, the above experiment was repeated with signal and noise being presented through a common sound source. With the use of conventional instrumentation, the advantages of binaural hearing aids cannot be demonstrated (Table 2). One may conclude that the signal and noise must be presented to the subject from different directions.

TABLE 2.—*Comparative Scores of Monaural and Binaural Hearing Aids with Signal and Noise Coming from a Common Transducer*

	Monaural		Binaural	
	Mean	Range	Mean	Range
Speech reception threshold	45.8 db.	44–48	43.6 db.	42–45
Discrimination in noise				
S/N +10	86.4%	80–90	86.8%	80–100
S/N 0	74.8%	68–78	66.0%	60–76
S/N −10	0.0%	0–0	0.0%	0–0

Summary

Discrimination scores with binaural hearing aids were not significantly different from those obtained with monaural hearing aids when the test words were presented at a level of 10 db. above the intensity of the noise.

Significantly better discrimination scores were obtained with binaural hearing aids when the test words were presented at the same intensity as the noise and at a level of 10 db. below the intensity of the noise.

Conventional clinical instrumentation does not permit demonstration of the superiority of binaural over monaural hearing aids. Signal and noise must be presented to the subject from different directions.

Comment

In the past, it has been generally concluded that the "ideal hearing aid candidate" is capable of functioning within normal limits when wearing a monaural hearing aid. The results of this clinical experiment suggest, however, that, when fitted with true binaural hearing aids the subject experiences significantly better discrimination when exposed to a listening environment which is strongly influenced by a competitive background noise.

First Ave. and 27th St. (16).

An Oto-Audiological Evaluation of Forty-Four Premature Children

PETER A. CAMPANELLI, M.A., Washington, D. C.; FREDERIC J. POLLOCK, M.D., and ROBERT HENNER, M.D., Chicago

During the past three decades, a number of clinical and statistical studies have been done on the prematurely born population. However, very little attention has been directed toward exploring the auditory sensory modality in this particular group of children. Previous emphasis has been directed toward investigating the effects of oxygen therapy during incubation of the premature neonate. Medical findings have described in great detail the serious visual impairment which occurs as a direct result of an overexposure of the infant to oxygen during incubation.[1-3] More recent studies have examined such interesting factors as neurological sequelae,[4] psychosocial adjustment,[5] and physical development [6] in this select pediatric population.

Johnsen's [7] results in Denmark, however, served to highlight the fact that a certain percentage of children in his study demonstrating hearing losses also presented a medical history of prematurity. For the most part, it was our feeling that this particular population had not been subjected to a thorough otological and audiological evaluation. This paper, therefore, is a preliminary report of such an investigation.

During the course of routine otological and audiological examinations, clinical attention was sharply focused on the occurrence of sensory-neural hearing impairments in a number of these premature children. It was speculated that this special segment of the pediatric population might possibly contain a greater incidence of auditory impairments than would be expected in a nonpremature population of a similar chronological age range. To test this premise, a research project was initiated with the cooperation of the Department of Pediatrics and the Premature Nursery Unit concerning the factor of hearing defects as related to either premature birth or concomitant postnatal management.

Selection of Cases

Several criteria were established for the selection of subjects for study in this project. These were in reference to birth weight, chronological age at time of examination, and otologic history. Specifically, these were as follows:

1. The definition of "prematurity" was based on the recognized pediatric reference of a birth weight of 2500 gm. (5 lb. 8 oz.) or less, regardless of the length of gestation. "Nonpremature" cases, therefore, had birth weights greater than 2500 gm.

2. Children were selected within the chronological age range of 72 to 84 months. On the basis of clinical experience, this age range was employed as the ideal minimum level to insure test reliability in the audiological evaluations.

3. No child was used as a subject who presented either a medical history or otologic evidence of an

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From the Hearing and Speech Service, and the Department of Ear, Nose, and Throat, Michael Reese Hospital Medical Center.

The main topics in this paper were read before the Sixth International Congress of Otolaryngology, Washington, D. C., May 8, 1957.

Dr. Pollock's present address: University of Illinois School of Medicine, Department of Otolaryngology, and Illinois Eye and Ear Infirmary, Chicago.

external or middle ear involvement. This was considered essential to minimize adventitious otologic disease which could confuse the audiologic results.

Two population samples were thereby established for study, namely, the premature or experimental group, and the nonpremature or control group.

The premature subjects were obtained through the nursery files of the Hospital. Parents of several hundred such children were contacted and their cooperation was requested in a special research project. No mention was made of the nature or intent of the study in order to minimize parental bias as much as possible. A total of 44 children was finally assembled, and this comprised an incidental sample of the premature population cared for in the Nursery between June, 1948 and June, 1949. An equal number of children was chosen from the various outpatient clinics of the Hospital, constituting an incidental sample of a "normal" control population. Each individual subject was selected according to the test criteria mentioned. Each group totaled 44 children, or 88 individual ears per group for examination.

In essence, an attempt was made to limit spurious effects as much as possible and to present the "prematurity" factor as the outstanding variable between two selected population samples.

Examination Procedure

Following the selection of the populations, each child underwent both a medical and a paramedical evaluation.

The medical examination consisted of a routine otolaryngological inspection prior to audiological measurements. This included information for a medical history, followed by an inspection of the oral, nasal, and pharyngeal structures, and otoscopic examination of the ears. Special attention was paid to the following: the oral cavity; condition of teeth; tonsils and adenoids; palatal arch; velopharyngeal closure; nasal airways; size and shape of the auricles; external meatuses, and condition of the tympanic membranes. When cerumen prevented complete visualization of the tympanic membranes, it was removed.

This examination determined the physical condition of the outer and middle ear structures. If pathology was present or suspected, the child was rejected for further participation in the study. On the other hand, negative findings allowed the child to be scheduled at a later date for an individual diagnostic evaluation of his communication skills, of which an audiological assessment was but a part. To insure the validity of these auditory measurements, the parents were instructed to cancel the appointment if their child showed actual evidence of, or recent recovery from, an upper respiratory infection. When this occurred, the child was merely rescheduled and tested when in good health.

The audiological assessments were completed within a two-month period after the medical examination. They were conducted in a sound-treated testing room having a relatively low level of ambient noise and employing a clinically calibrated audiometer. Air-conduction threshold measurements were obtained for the test frequencies 125 through 8000 cps in octave steps, as well as bone-conduction threshold measurements for the frequencies 250 through 4000 cps in octave steps. Suitable precautions were employed during the test procedure to insure reliable responses. The method of limits was employed in determining the threshold levels. As a necessary supplement, speech reception thresholds were obtained both monaurally and binaurally by means of monitored spondaic words. Speech discrimination scores were also obtained using the monitored PB 50 word lists. These verbal materials were presented by means of the recorded C. I. D. adaptation of the Harvard PAL lists W 1 and W 22.

As previously mentioned, additional areas were also evaluated along with the audiological assessment. These areas included (1) examination of the oral and speech musculature; (2) locomotor coordination; (3) laterality; (4) auditory memory span for digits; (5) expressive and receptive language usage; (6) speech articulation, and (7) phonation. The results of these performances need not be mentioned here, and will be reported on in subsequent papers.

Each subject, both premature and control, underwent the following examination procedure: (1) medical case history; (2) routine otolaryngological examination; (3) audiological assessment.

Test criteria were scrupulously adhered to so as to prevent or minimize those conditions which would tend to obscure the final results.

Results

Taking the two population samples under consideration, it will be remembered that

TABLE 1.—*A General Descriptive Mean Analysis of Forty-four Premature and Forty-four Normal Children Comprising the Population Samples*

	Premature	Normal
Sex		
Male	15	24
Female	29	20
Race		
Negro	39	38
White	5	6
Gestation, wk.	32.6	39.3
Birth Weight, gm.	1648	2978
Chronological age, yr.-mo.	6-4	6-6

there was an N-44 per group. A general mean analysis of the two groups is presented in Table 1 with regard to the sex and racial distribution, period of gestation in weeks, birth weight in grams, and the chronological age in years and months at the time of the examination.

Several items should be noted in Table 1. Interestingly enough, there were twice as many females as males in the premature group, there being a closer distribution in the control group. This may be due to the fact that the birth proportion as well as a lower mortality rate favors the female, thus allowing for a greater number in the test sample. In the racial grouping of the subjects, it should be pointed out that the unusually high incidence of Negro cases in both groups is in keeping with the racial distribution of the population served by the outpatient clinics. Further, the chronological age refers to the mean age at the time of examination. The data on gestation and birth weight were taken from the Hospital medical records.

For both groups, the otolaryngological findings were essentially negative with respect to ear, nose, and throat physical findings. All subjects were considered acceptable, from the otological criterion, for participation in the audiological procedures. Regarding the children in both groups, no individual was eliminated because of any outer or middle ear pathology. On the basis of the results of this medical examination, it was the impression that if any auditory impairment did exist in the premature group, it was certainly cochlear or retrocochlear (i. e., sensory-neural) with respect to site of lesion.

Considering the audiological measurements on these children, some rather interesting results were noted. The mean threshold measurements, as well as speech scores, for the control group of 44 children are depicted in Figure 1. As would be expected, the mean auditory acuity is well within normal limits for both pure tone and speech stimuli. Standard deviations for all measures (air and bone conduction, speech reception and speech discrimination) in this control group were extremely small, the range being between 2.6 and 6.1. These audiological findings revealed no incidence

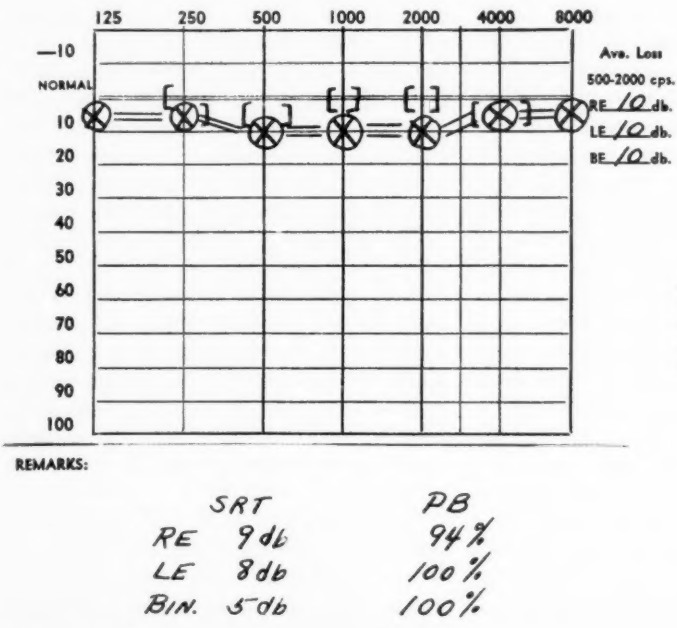

Fig. 1.—Mean threshold measurements for air and bone conduction, and mean speech results, for control group of 44 non-premature children.

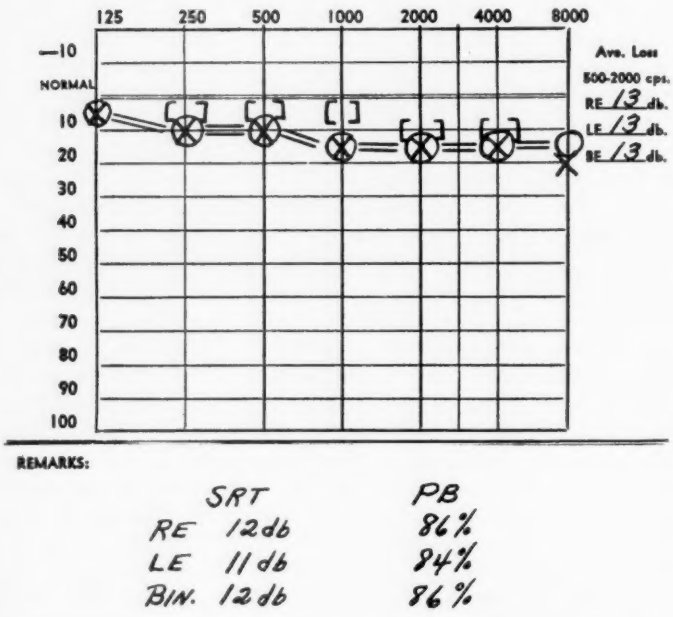

Fig. 2.—Mean threshold measurements for air and bone conduction, and mean speech results, for experimental group of 44 premature children.

of hearing loss in this control population of 44 children with a history of nonpremature birth.

The mean threshold measurements, as well as speech scores for the experimental group of 44 premature children, are shown in Figure 2. With reference to the previous control group, these measurements do not appear significantly different, except in one respect, that is, this group demonstrates a reduced ability in speech discrimination as shown by the binaural P B score of 86%. However, this difference is explained by pointing out that this score represents a mean group measure which obscures two important items. The first is the extremely large standard deviations around the mean threshold findings. For example, the range is from 11.2 to 26.0 for air-conduction means, 4.5 to 16.2 for bone-conduction means. Similarly large deviations are also found in the speech scores. Most important, however, is that such deviations are consistent with the second observation of the presence of a number of hearing losses in this particular sample. It may be stated that in this experimental group of 44 premature children, 7 (15.9%) demonstrated definite hearing impairments, ranging from moderate to severe, and presumably sensory-neural in type.

This factor is more apparent when the mean results of these seven cases are abstracted and presented in Figure 3. It is observed that this audiometric configuration for both air and bone conduction is typical of a sensory-neural involvement. The extremely severe reduction in discrimination is felt to be a result of the severer losses coupled with the greater losses for the high-frequency tones. With these results in mind, the observation is made that there appears to be an incidence of hearing impairment in the premature population under study to the extent of 16% of the total group. Further, this defect appears to be a bilateral involvement, extending from moderate to severe regarding degree of involvement, and sensory-neural with respect to site of lesion.

When these significant audiological findings were presented, an attempt was made by retrospect observation to explain various factors relative to ascribing etiology. A

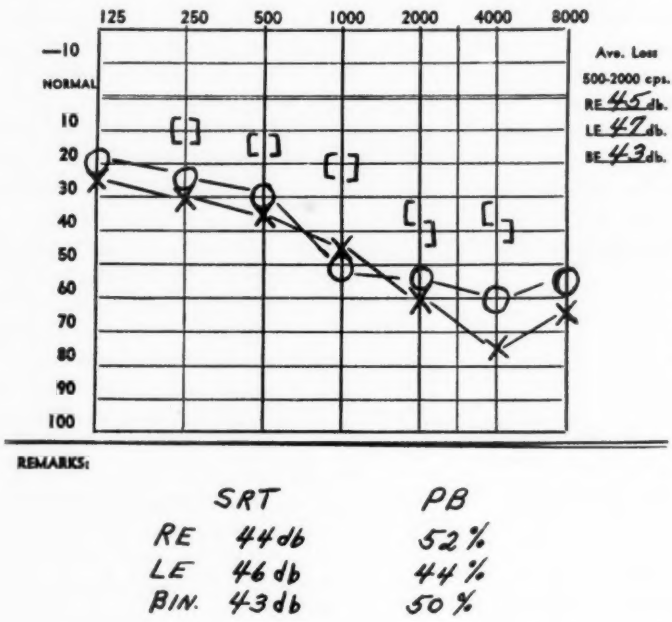

Fig. 3.—Mean threshold measurements for air and bone conduction, and mean speech results, for seven premature cases demonstrating hearing losses.

methodical examination of the prenatal histories of the mothers of these seven cases failed to uncover any maternal illness or condition which might cast suspicion on a possible cause-and-effect relationship regarding the auditory defect. Perinatal reports were also negative. Postnatal findings regarding illnesses, diseases, medication, etc., were not out of the ordinary with regard to type and degree.

Several specific items, however, were statistically analyzed. These were (1) period of gestation; (2) duration of labor; (3) birth weight, and (4) duration of incubation. That is to say, the 7 premature cases with hearing losses were contrasted with the remaining 37 premature cases without hearing losses, and the mean results are depicted in Table 2. In this table are listed those items which are pertinent to the premature group. "Incubation" refers to the time, in days, spent in a special apparatus for maintaining optimal conditions (temperature, humidity, etc.) following parturition. The term "Conc. O_2" refers to the concentration of oxygen maintained during the period of incubation.

A statistical comparison regarding the significance or nonsignificance of the difference between the group means is also noted in Table 2. It should be pointed out that the concentration level of oxygen was always maintained at 2-3 liters. This was due to the fact that prior to 1952 no accurate percentage level of oxygen was thought necessary. For this reason, no statistical measure could explore this variable. However, in those items where comparison was possible, there is a difference between the group means regarding length

TABLE 2.—*A Mean Comparison of Seven Premature Cases with Hearing Losses and Thirty-seven Premature Cases Without Hearing Losses; Levels of Significance also Included*

	7 Premature	37 Premature	Lev. of Sig.
Sex			
Male	3	12	
Female	4	25	
Race			
Negro	4	35	
White	3	2	
Gestation, wk.	26.3	33	--
Labor, hr.	4	4	--
Birth Weight, gm.	981	1718	0.05
Incubation, days	24	8	0.01
Conc. O_2, liters	2-3	2-3	

of incubation significant at the 1% level, and a difference significant at the 5% level with regard to the group mean for birth weight. There was no statistical difference between the two groups regarding the period of gestation or duration of labor. It would appear, therefore, that the factor of birth weight and length of incubation in this particular sample are significant as possible causative factors.

Comment

It is interesting to speculate on possible causes and effects with regard to the demonstrated hearing defects. It might be theorized that the gestation period, shortened or otherwise, resulted in an extremely debilitated neonate (i. e., low birth weight) requiring a long-term postnatal management, specifically, incubation. Whether or not the oxygen factor is an important variable cannot be stated at this time, although it has been shown previously that an overabundance may cause visual impairments. In our experience, however, we have not seen any blind premature children as a result of retrolental fibroplasia who were also hard of hearing or deaf. Further research on premature children born after 1952 should prove fruitful since there has been better control of oxygen therapy.

From the findings reported here we are unable to ascribe to any one single agent responsibility for the auditory defect, but certainly a low birth weight and a long duration of incubation must be considered closely related to the condition. Previous mention of Johnsen's[7] work in this regard offers valuable insight into the probable etiology of the sensory-neural hearing loss. In his study of 39 children with perceptive deafness, his analysis indicated that 9, or 23%, were premature. His study of the natal cause of deafness in this premature group turned up a high incidence of icterus neonatorum, hemorrhage, and anoxia. This is in contradistinction to the present results, wherein no medical notation of these conditions was found. These factors noted by Johnsen may, in themselves, be predisposing causes for auditory difficulties even without prematurity. The experiences reported in this paper, however, would tend to support Johnsen's belief that perhaps both prematurity and sensory-neural lesions are brought about by the same obscure noxious agents.

The findings reported in this paper should be considered significant and clinically valuable, even though further study is indicated in the area of premature children regarding the auditory function. These results (i. e., that 16% of this particular group displayed a hearing defect) should serve to alert the clinical worker to suspect the presence of a sensory-neural hearing loss whenever a premature child is examined, particularly when there has been a history of a very low birth weight and a long period of incubation. In other words, it might be well to suspect every premature child of having a hearing impairment until a thorough otological and audiological evaluation proves otherwise.

It may be of interest to note here that similar results pertaining to this problem were brought to our attention by Cubert.[8] Her analysis of clinic records of 145 consecutive pediatric cases having sensory-neural hearing losses revealed that 22 cases, or 15.1%, of the total group were prematurely born.

Continued research is indicated to explore the intriguing possibilities inherent in this particular group of children with regard to the hearing mechanism, in view of the fact that prematurity accounts for roughly 8% of the total live birth population.

Summary and Conclusions

This preliminary report is an oto-audiological evaulation of the hearing mechanism in a population of 88 children in the age range of 72 to 84 months, equally divided into a premature and a control group. Test criteria were employed for the selection of cases and the following results are reported:

Otolaryngological findings were essentially negative in both groups regarding external and middle ear pathology.

Audiological measurements conducted on the 44 children in the control group indicated that results were within normal limits for both pure tone and speech stimuli.

The sample of 44 premature children tested displayed 7 cases (15.9%) with a bilateral, sensory-neural hearing impairment, extending from moderate to severe in degree of loss.

Statistical analysis of various items in the medical history of these seven cases casts significance on the factors of the birth weight and length of incubation as possible causative factors.

Continued investigation is indicated to explore the possibilities inherent in this particular pediatric population.

Ralph Kundstadter, M.D., and Evelyn Lundeen, R.N., of the Department of Pediatrics and the Premature Nursery Unit aided in this study.

District of Columbia Department of Public Health, Washington (Mr. Campanelli).

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Case Reports

Simple Fibroma of the Nasal Septum

M. VALENTINE MILLER, M.D., Philadelphia

Benign growths of the nasal septum are rare and include granulomas and vascular tumors, which seem to be more frequently found than chondromas, myomas, and fibromas. Except for the bleeding which may occur at times from granulomas and vascular tumors, such as angiomas and telangiectatic polypi, these benign tumors usually cause no symptoms other than nasal obstruction and, if allowed to develop so far, deformity of the nose and face on the side involved. The etiological factor in the development of these benign tumors is not clear, though it seems reasonable to assume that in many of them trauma from whatever source, whether physical trauma or irritation from chronic sinus involvement or inhaling some chemical or dust irritant, may be the trigger which starts the growth.

The case here reported is one of simple fibroma of the nasal septum. A search of the literature has revealed only two such cases prior to this one. In one of the cases already reported by Lins e Silva [1] there was a history of trauma 17 years before the patient was first seen. In neither the case which is being reported here nor the one reported by Simpson and Evans [2] was there anything in the history to even suggest the etiology. Both cases previously reported occurred in women, one [1] aged 42 and the other age not stated. The case here reported was in a man who was 79 when first seen.

As stated before, the only subjective symptom in any of these three cases of fibroma of the septum was that of nasal obstruction, and it was this alone which caused all three to seek relief. As to objective symptoms, all three showed a mass arising from the quadrilateral cartilage; all three tumors were rounded and smooth on the surface; all were covered with mucosa which was similar to the rest of the nasal mucosa; all three were firm and noncompressible; one had a fairly wide pedicle, while the other two were sessile; one patient [2] gave a history of "slight bleeding," while there was no bleeding at any time from the other two; the case here reported had marked deformity of the face and nose, while the other two showed no deformity.

As with all nasal tumors, I feel that a biopsy should be done before the type of surgery is decided upon, as one cannot determine from the gross appearence whether one is dealing with a fibroma, a chondroma, or a chondrosarcoma. When one is sure he is dealing with a fibroma, the best method of removal would seem to be the undermining of the perichondrium and removal of the entire mass. In one of the cases already reported [2] the whole mass, including the overlying mucosa and the underlying cartilage, was removed, after which a sliding flap was made of the mucosa remaining on that side and this drawn over the denuded area. In the other case [1] the mucosa was incised over the mass and separated from it; the perichondrium underlying the mass was separated from the cartilage, and the whole mass removed. After this the redundant mucosa was removed and the wound closed, leaving the cartilage in place. In the case being reported, the patient, who was 79 years of age, after the major portion of the mass had been removed for biopsy considered the relief so great that he repeatedly refused to have further surgery. He

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has promised he will submit to the total removal should his obstruction recur. In the meanwhile he has promised to return at intervals for check-up.

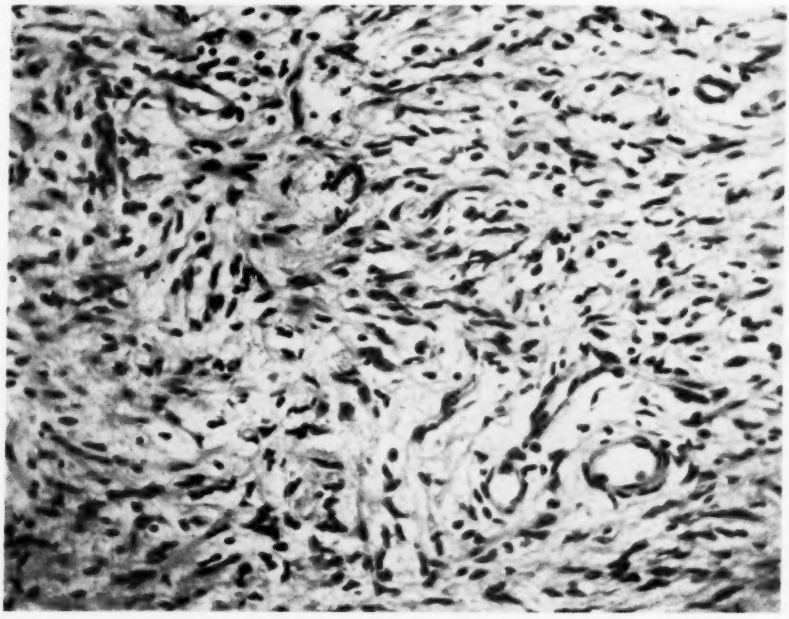
Report of Case

A white man aged 79, a retired building supervisor, came to the office to inquire about a mass in his right nostril which markedly interfered with his breathing and in addition had caused a rather marked bulge on the right side of his nose and adjacent maxilla. He could get some air through his nose on the right but it was little. He stated that he had had the external deformity for about one year and did not feel that it had increased. He could not recall when the nasal obstruction had begun, but it seems obvious that this was of long standing and that the external deformity must have been present for a considerably longer period than one year since he was not aware of any increase in the deformity since he first noted it. On rare occasions, and only after a severe sneeze he has noted a few streaks of blood in the nasal secretions. He sneezed "a good bit," as he put it, but not in bouts, and there was no personal or family history of allergy. He said he rarely had any nasal or postnasal discharge. He has had few colds or upper respiratory involvement of any type. There was no history of trauma, chemical or physical. He had never had any ear or throat symptoms at any time, and the rest of history was unimportant.

On examination there was a bulging outward of the nasal bones on the right and also of the cartilaginous portion of the nose. There also appeared to be some bulging of the maxilla adjacent to this. There was no tenderness over the area. On intranasal examination there was a large, smooth, very firm and fixed mass which appeared to fill up the entire right side of the nose, with the exception of a narrow airway between it and the floor. The color of the covering membrane was the same as the rest of the nasal mucosa, though possibly a shade paler. A nasoscope was introduced with some difficulty under the mass and the latter was found to be attached by a broad base which appeared to be almost completely attached to the cartilage but possibly had a short extension over on the perpendicular plate of the ethmoid. The rest of the intranasal examination showed no other pathology. Nasal cytology was normal. Transillumination of the frontal and maxillary areas was clear and even on the two sides. All teeth had been extracted, and the tonsils were atrophic. Mirror examination of the larynx showed no pathology. There were no palpable cervical nodes.

It was decided to remove a portion of the tumor for histological examination, and under topically applied tetracaine (Pontocaine) 2% and epinephrine the mass was removed with a snare and with a minimal amount of bleeding. Grossly the

Photomicrograph of fibroma of nasal septum.



cut section was almost white, appeared homogeneous, and was very firm. After the removal, that side of the nose was packed. The packing was removed the next day, and there was a small amount of bleeding which was readily controlled. Some of the base still remained, but the patient was allowed to return home to await the pathological report. There was very little postoperative discomfort.

The pathological report * was as follows: "Gross Examination: The specimen consists of a flat, oval mass measuring 2.5 cm.×2 cm.×1 cm. The surface is smooth and of a light tan color (it had been in preserving fluid). On section it is composed of a homogeneous light tan, translucent tissue. Microscopic Examination: of the mass shows that it is covered in part by a stratified squamous epithelium and in part by columnar epithelium. The stroma of the mass is composed of a loosely arranged fibroconnective tissue. The fibrils are fine and separated. There are occasional focal connections of lymphocytes and plasma cells. Diagnosis: Fibrous polyp of the nasal septum."

When the patient reported three days later he said he had had no bleeding or discomfort and he was breathing as he had not breathed for many months. As the pathological report showed that the tumor was benign, he flatly refused to have anything further done, saying that the condition had never bothered him except for the obstruction, and he had come only because he had been urged by his family physician; he could not see the need for any further surgery as he was breathing so well since the main mass was removed and found to be benign.

Some time later he was called on the phone and requested to return for a check-up, and a little less than three months after the removal he finally appeared. He was still breathing freely and had no discomfort. However, on examination there appeared to be some slight increase in the bulk of the base which remained, particularly above, and in the center of this was a small mass about the size of a matchhead which bled when touched with an applicator. This appeared to be granulation tissue, but it was removed for histologic examination, and the base of it was touched with an electrocoagulating electrode. The histologic examination showed it to be granulation tissue.

The patient still refused further surgery but promised to return from time to time for a check-up. There had been some improvement in the external deformity of the lower part of the nose, but the nasal bones still were as they had been when first seen.

114 W. Phil-Ellena St. (19).

* Pathological reports by Dr. F. E. Haentze, Pathologist to Roxborough Memorial Hospital, Philadelphia.

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Rhinomucormycosis Resulting in Fatal Cerebral Mucormycosis

GREGORY K. DWYER, M.D., Norwalk, Conn., and GEORGE W. CHANGUS, M.D., Burlington, Iowa

The early identification of the primitive class of fungi, the Phycomycetes, in the biopsy and culture of the nasal and paranasal lesions of two diabetic patients led to the control of the spread of the fungi into the brain, where fatal cerebral mucormycosis might have resulted (Harris [1]; Baker, Andrews and Izlar [2]). The fungi of the genus Rhizopus of the order Mucorales were identified in both patients. Fifteen of the seventeen recorded cases, including the present case, died as a result of the spread of the fungi into the brain (Table). Earlier recognition through biopsy or culture of the nasal or paranasal lesions may have averted cerebral spread and death.

All of the reported cases had an underlying, debilitating disease. Uncontrolled or poorly controlled diabetes mellitus [1-7] provided a favorable medium of growth of the fungi in 11 of the 17 recorded cases, including the present case. The underlying diseases in the remaining six cases were nephritis [8,9] in two cases, dehydration and marasmus.[10]

Submitted for publication Dec. 6, 1957.

From the Departments of Otolaryngology and Pathology, Norwalk Hospital.

Permission to report this case was given by Dr. George W. Hebard, New Canaan, Conn.

Assistant Clinical Professor, Department of Otolaryngology, Yale University School of Medicine (Dr. Dwyer). Present address of Dr. Changus: Director of Laboratories, Mercy Hospital, Burlington, Iowa.

Summary of Cases of Rhinomucormycosis

Author	No.	Age	Race	Sex	Route of Spread: Nasal, Paranasal	Route of Spread: Perior-bital	Route of Spread: Cerebral Vessels	Route of Spread: Brain	Associated Disease	Days of Survival
Gregory, Golden and Haymaker, 1943	1	43	N	F	+	++	++	++	Diabetes	2–4
	2	52	N	F	++	++	++	++	Diabetes	5
	3	75	W	M	?	++	++	++	Diabetes	2
LeCompte and Meissner, 1947	4	57	W	M	?	+	++	++	Diabetes	2
Wolf and Cronin, 1949	5	42	N	M	+	+	++	++	Diabetes	17
Stratmeier, 1950	6	32	N	F	++	++	++	++	Diabetes	2
Kurrein, 1954	7	5 mo.	W	M	+	?	++	++	Nephritis	few
Martin, Lukeman, Ranson, and Gippert, 1954	8	2.5 mo.	W	M	?	++	++	++	Dehydration; marasmus	14
Bauer, Ajello, and Adams, 1955	9	25	W	F	—	+	++	++	Diabetes	7
	10	40	N	M	+++	++	++	++	Diabetes	6
Harris, 1955	11	14	N	F	+++	+	—	—	Diabetes	Living
Gunson and Bowden, 1956	12	8	W	F	?	?	++	++	Nephritis	10
Foushee and Beck, 1956	13	65	W	F	+	++	++	++	Liver abscess	12
Mayfield and Condie, 1957	14	24	W	M	+	+	++	++	Leukemia	?
Baker, 1957	15	62	W	M	++	+	++	++	Liver cirrhosis	14
Baker, Andrews, and Izlar, 1957	16	Adult			+++	++	—	—	Diabetes	Living
Dwyer and Changus, 1957	17	46	W	F	++	++	++	++	Diabetes	9

? not investigated or unknown.
\+ clinical or gross evidence.
++ confirmed by sections of tissue.
+++ confirmed by sections and culture (all cultures to date of genus Rhizopus).
— negative clinical or gross evidence.

in one case, liver abscesses following cholecystectomy [11] in one case, leukemia under 6-mercaptopurine and cortisone therapy [12] in one case, and, cirrhosis of the liver under penicillin therapy [13] in one case. The experimental studies of Bauer, Flanagan, and Sheldon [14] indicate that the altered polymorphonuclear leukocytic function induced by the disturbed metabolism of the underlying disease may be an important factor in the pathogenesis of the mycotic infection.

The main purpose of reporting this case is to emphasize the nasal and paranasal route of spread of the fungus, so that earlier identification of the fungus will lead to the control of its spread to the brain. The designation of rhinomucormycosis is, likewise, introduced to emphasize the controllable portion of this entity rather than the uncontrollable cerebral mucormycosis.

Report of a Case

A 46-year-old white woman was admitted to the hospital on Nov. 1, 1955, because of an acute left ophthalmoplegia and lid drop. Three days prior to admission she had noted throbbing pain in the left upper jaw area which had become severer, with a feeling of numbness and swelling in the left cheek. She was edentate. The morning of admission to the hospital the pain became intense in the entire left side of her face and left temporal area.

Past History.—About four years before the present illness she had pneumonia, and at that time it was found that she had diabetes mellitus which required insulin and diet control. It was difficult to control her diabetes because of her negligence and alcoholic intake.

About three years before the present illness she was admitted into the hospital because of extreme weakness, anorexia, chills and fever, which were found to be due to a right renal necrotizing papillitis and multiple abscesses of the right kidney. After removal of her right kidney, she slowly recovered, and six weeks later was discharged from the hospital on insulin and diet control.

Interval History.—The three-year interval period from Dec. 11, 1952, to the terminal admission on Nov. 1, 1955, was not remarkable except that her diabetic control was poor and she continued to consume alcohol.

Physical Examination.—The significant physical findings on Nov. 1, 1955, were confined to the left side of the face and head. There was complete left ophthalmoplegia with lid drop, absent corneal reflex, dilated pupil that was fixed to light and accommodation, slight blurred vision, normal ocular tension, no ocular tenderness, normal fundus, and hyperesthesia over the ophthalmic division of the left fifth nerve. The oral temperature was 100 F. The impression was intracranial vascular lesion or possible aneurysm.

Hospital Course.—The following day, on Nov. 2, facial edema was noted. The fasting blood sugar was 354 mg. per 100 ml., and the urine had 4+ sugar, 2+ acetone, 4+ albumin, and 20-25 white blood cells per high-power field. She was started on insulin and diet control. The white blood count was 16,100 per cu. mm., with 83% adult polymorphonuclears, 9% stab forms, and 8% lymphocytes.

On Nov. 3 the facial edema increased; there was complete blindness of the left eye, peripheral palsy of the left seventh nerve, and the temperature went up to 103 F. One of us (G. K. D.) was consulted and found the patient to have complete left facial palsy, exquisite tenderness over the left jugular, a foul, yellow pus filling the left nasal vault, a dry, foul, hyposensitive palate, and sinuses that were dark to transmissible light on the left. The left eardrum was dull, but the hearing normal with tuning fork. The impression was that the patient had an acute cavernous sinus thrombosis with involvement of the left jugular bulb secondary to acute fulminating left pansinusitis which was predisposed by the diabetic acidosis. The spinal fluid was slightly cloudy and had 277 white blood cells, of which 67% were polymorphonuclear leukocytes and 33% lymphocytes. The spinal fluid proteins were 50 mg. per 100 cc., glucose 284 mg. per 100 cc., chlorides 124 mEq. per liter, and the bacterial culture was negative. A blood culture was negative for bacteria. Hemolytic Staphylococcus aureus var. albus (Micrococcus albus) was cultured from the nose that was mannitol-positive, coagulase-positive, highly sensitive to tetracycline (Achromycin) and moderately sensitive to penicillin. She was given 100,000 units of penicillin, intrathecally; 300,000 units of crystalline penicillin every four hours, intramuscularly, and 500 mg. of tetracycline every six hours. The left antrum and frontal were irrigated through the natural ostia, and a copious quantity of thick yellow pus was obtained. Heparin and bishydroxycoumarin (Dicumarol) were given, but the heparin was discontinued the following day because of vaginal bleeding in the midmenstrual period. On Nov. 4, one of us (G. K. D.) noted after irrigation of the antrum that there was a foul, gelatinous, dirty material in the washings, and a black-gray eschar covered the left hard palate. A direct smear for fungi was suggested, but, unfortunately, the suggestion was overlooked. A roentgenogram of the sinuses

was interpreted as opacifications of the left maxillary sinus and ethmoid air cells, probably due to acute sinusitis.

Her condition became worse. The temperature rose above 104 F; the facial edema increased, and the cranial nerve involvement from the second to the seventh inclusive was complete on the left. No right-side involvement was present. There was tenderness of both globes but more on the right than on the left. Four antibiotics, including penicillin, tetracycline, streptomycin, and sulfadiazine, did not seem to benefit the patient.

On Nov. 6 (the fifth hospital day) she lapsed into coma and had convulsive twitchings of the left arm. The left periorbital skin areas and the left side of the nose were cold and gangrenous. There was ptosis of the right lid and ophthalmoplegia of the right eye for the first time. The left eardrum now showed a pulsating perforation, through which a serosanguineous fluid drained. The possibility of a radical approach through the left mastoid for drainage was considered, but the elevated prothrombin time and her poor condition precluded the attempt. Early the following morning, at 5:15 a. m., on Nov. 7, 1955, the patient died, six days after admission to the hospital and nine days after the onset of the terminal disease.

Autopsy Findings.—There was moderate bulging and cyanotic discoloration of the left side of head, more marked in the lids and periorbital tissues. There was gangrene with superficial ulceration of the skin in the left infraorbital region. The left naris was gangrenous and contained a hemorrhagic, gray-red, soft exudate. The left upper lip and cheek were swollen, cyanotic, and mottled with petechial hemorrhages. The left eye was markedly icteric and was filmed with a hemorrhagic, gray-red, soft exudate. A similar exudate was present in the left, external auditory canal.

There was a layer of soft, gray-red exudate over the frontal portion of the temporal lobe of the brain. A 5 by 5 by 3 cm. portion of the anterior left temporal lobe was very soft and gray-red. The meninges of the brain were injected, especially on the left side. The left cavernous sinus was distended with a brown-red, firm, and friable clot which was adherent to the wall. The left sigmoid sinus contained dark red, clotted blood, which was mottled with soft, yellow-white particles. The right cavernous sinus had a moderately firm, yellow-white clot which was adherent to the wall. Aside from moderate congestion of the vessels the remainder of the brain was intact.

The left ethmoid and maxillary sinuses contained inspissated, yellow-white fragments. The surrounding bone contained brown, necrotic soft tissue. The adjacent muscle tissue was tan and necrotic. There was similar necrosis of the petrous portion of the left temporal bone. The mastoid sinuses were intact. The periorbital tissues as well as the optic nerve were involved with gray-red soft material. The maxillary nerve and sphenopalatine ganglion on the left had similar gray-red material.

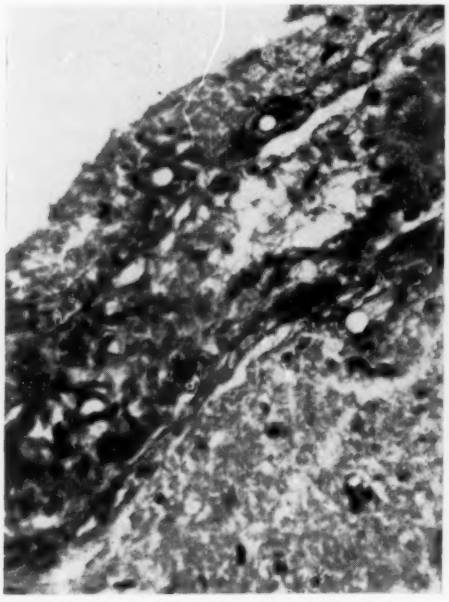

Figure 1

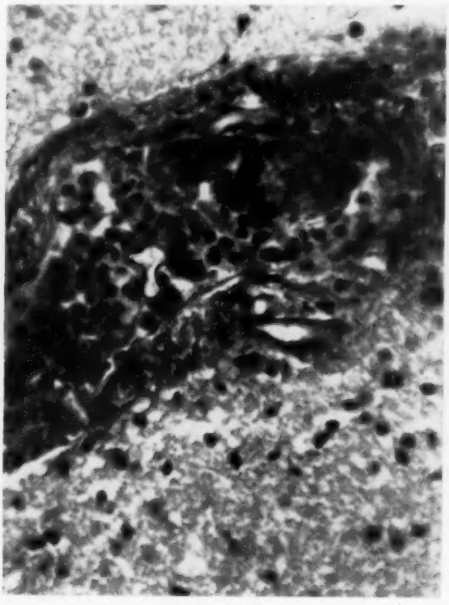

Figure 2

Figure 3

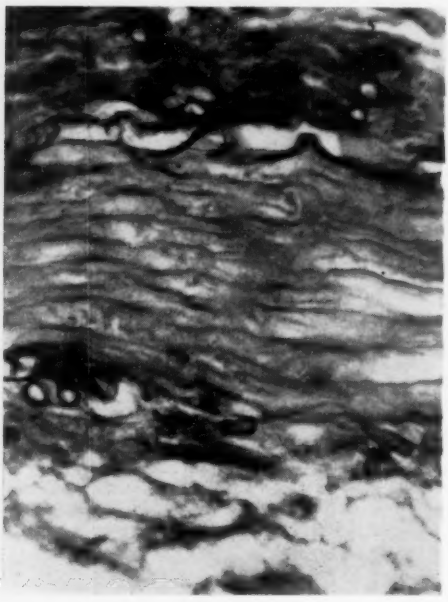

Figure 4

All the cultures of the above involved areas were negative for bacteria, except the left sigmoid sinus, which had hemolytic Staphylococcus aureus var. albus that was coagulase and mannitol-negative. Fungal cultures were, unfortunately, not taken.

The remainder of the autopsy disclosed a fatty liver that was enlarged one and one-half times normal size, an absent right kidney, slight hydronephrosis and chronic pyelonephritis of the left kidney, and moderately congested lungs.

Microscopic Findings.—Although there was a marked polymorphonuclear reaction in the necrotic tissue of the left maxillary sinus, meninges (Fig. 3), and brain (Fig. 4) with an admixture of branching, irregular fungi mainly confined to the thrombi and walls of vessels, the reaction to the fungi themselves was negligible, as seen in the perineural vessels (Fig. 1) and palatine bone (Fig. 2). The leukocytic reaction was associated with infarcted areas induced by the vasculophilic, thrombus-provoking fungi. The route of spread of the fungus was demonstrated from the maxillary sinus into adjacent bone, along vessels, especially through the perineural vessels of the branches of the maxillary nerve, into the spenopalatine ganglion involving the periorbital tissues, both cavernous sinuses, meninges, and brain. The fungus was considered identical with that described in tissue sections and cultured by Bauer, Ajello, and Adams,[7] Harris,[1] and by Baker, Andrews and Izlar,[2] or at least belonging to the order Mucorales, if not the genus Rhizopus.

The remainder of the microscopic findings confirmed the gross findings of a moderately fatty liver, congested lungs and a moderately hydronephrotic kidney with severe chronic pyelonephritis.

Comment

The route of spread of this fungus by the way of the nasal and paranasal tissues, via the blood vessels, especially about nerves, into the periorbital tissues and venous sinuses and into the brain, has been demonstrated in the present case. A similar route of spread has been noted in nearly all the previously recorded cases, with questionable exception of five cases (Table). Early recognition of the nasal and paranasal phase of this entity, especially by the consulting otolaryngologists, will perhaps lead to control of the fungus and increase the salvage of these unfortunate patients by the restoration of their disturbed metabolism, reevaluation of the bacterial antibiotic or drug therapy, and the institution of antimucormycotic agents.

As can be seen from the Table, the course of this disease is extremely fulminant and short, ranging from 2 to 17 days in the fatal cases. Early recognition of the fungus in smears or biopsy of the nasal and paranasal lesions is required to make it possible to prevent the spread of the fungi to the brain. Treatment should begin as soon as smear or biopsy evidence is afforded. Confirmatory cultural identification should be done but it is not needed for the inception of treatment.

Summary and Conclusions

The route of spread of this vasculophilic and thrombus-provoking fungus of the order Mucorales from the nasal and paranasal tissues into the brain is demonstrated. A similar route of spread is noted in most of the recorded cases. Earlier recognition of the fungus in the nasal and paranasal lesions of debilitated or diabetic patients through smears and biopsy is urged. Control of the curable nasal and paranasal phase of this entity is emphasized. A new designation of rhinomucormycosis is introduced to describe the curable phase of this disease in contrast to the lethal phase known as cerebral mucormycosis. The pathogenesis of this entity is briefly discussed.

85 East Ave. (Dr. Dwyer).

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Buckshot Wound of a Vocal Cord

ROBERT Z. BERRY, B.A., M.D., and DONALD S. CARTER, M.D., Baltimore

Gunshot wounds of the neck are rather common, especially buckshot wounds as result of hunting accidents. Most buckshot injuries result in the shot being lodged in the cervical soft tissues and, except for pain, are relatively asymptomatic. Unless easily palpable, it is many time fruitless to search the soft tissues in hopes of removal of the shot. Small foreign bodies such as this are notoriously evasive to the surgeon's search. The following case is one in which a single "shot" entered the neck, lodged in the larynx, and was successfully extracted via the endoral route.

Report of Case

A 41-year-old white male patient was transferred to our hospital from another city after a "gunshot" wound of the neck. When first seen, the patient appeared in only minimal discomfort and was able to give the history that while hunting rabbits earlier that day, his hunting companion fired his shotgun at a fleeing rabbit. Although the direction of the shot was at a tangent to their position, a single pellet ricocheted, and the patient immediately felt a sharp pain in the front of his neck and when he attempted to notify his friend of the occurrence, he noted a rather marked hoarseness. The pain subsided quickly except for a slight ache. The hoarseness persisted until he was seen by us. There was no cough, hemoptysis, or shortness of breath.

Physical Examination.—When seen by us some eight hours after the injury, the patient was comfortable and asymptomatic except for hoarseness. External examination of the neck showed a single blood clot designating the site of entry of the shot in the midline of the neck in close proximity to the thyroid notch. There was only slight tenderness over the area, and no demonstrable swelling or crepitus. There was no discoloration. Mirror

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Figure 1

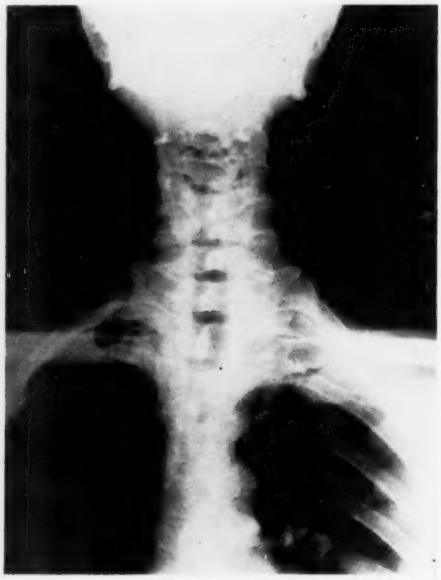

Figure 2

examination of the larynx revealed nothing abnormal except for an ecchymotic, edematous, immobile left true cord. The glottic breathing space was adequate. The remainder of the general physical examination was negative.

Laboratory Findings.—X-rays of the neck for soft tissue detail revealed a radiopaque foreign body compatible in size and shape with a buckshot, lying in the midline of the neck at the junction of the middle and lower ⅓ of the thyroid cartilage within the confines of the larynx. There was no evidence by x-ray of any subcutaneous emphysema. Urine analysis and blood count were normal.

Hospital Course.—The patient was given penicillin, tetanus antitoxin, and chymotrypsin (5000 units I. M.) on admission and put to bed. The following morning the patient was anesthetized by intravenous thiopental sodium and fractional dose of tubocurarine and succinylcholine. The larynx was visualized through a Lynch suspension laryngoscope. The shot could not be palpated in the cord, nor could it be localized with a magnet. A shallow longitudinal incision was made over the superior aspect of the left cord and the area gently probed with a small laryngeal alligator clamp. After a short interval the shot was palpated, grasped, and removed. Postoperatively the patient did well and was discharged from the hospital to return to his home in another city. The patient has been seen by us several times, and the voice has completely returned to its normal tone.

Dept. of Otolaryngology, Mercy Hospital (2).

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Cholesteatoma-like Accumulations in the External Auditory Meatus

Report of One Case

COL. BYRON G. McKIBBEN (MC), U. S. Army

Cholesteatoma-like accumulations in the external auditory meatus are not common. Scientific articles on this subject occasionally have appeared in medical literature. Different authors have referred to this condition as "molluscous tumors,"[1,2] "sebaceous tumors,"[3] "keratosis obturans,"[4,5] "pearl tumors,"[6] "epidermis plugs,"[7] "cholesteatoma,"[7,8] "cholesteatoma-like accumulations,"[9] "myringitis desquamativa,"[10] and "otitis desquamativa externa."[10]

The relative paucity of articles on this subject, published in English, appears to warrant the addition of the following case.

Report of Case

A 23-year-old Negro soldier was admitted to Tokyo U. S. Army Hospital, April 12, 1957, because of earache and purulent drainage from the left ear.

History disclosed that approximately eight years prior to admission he had had a full sensation and deafness in the left ear and that he had had intermittent earache for a period of about two years. Following this the only symptom he had, relative to the ear, was deafness, until three weeks prior to admission. At that time the left ear began to ache. About 10 days later the left ear began to drain, and the pain became less severe but never completely subsided.

On physical examination, the left external auditory meatus was occluded, and a polyp-like mass was presenting in the cavum conchae. Tuning fork tests indicated a conductive type deafness. Audiometry was not performed.

Under local anesthesia (1% procaine infiltration), the polyp-like tissue was removed with biting forceps. This revealed cholesteatoma-like material, which was removed in several small pieces and one large mass, measuring 3.0×1.5×0.5 cm. The large piece was almost a complete cast of the external ear canal. Thin, grayish-white pus was released as the cholesteatoma-like material was removed. When this procedure was finished, the patient exclaimed, "I sure am glad to get rid of that. That's the best I've been able to hear in eight years." The skin of the canal was hyperemic, and in some areas the skin was replaced by granulation tissue. The tympanic membrane was not clearly defined.

Grossly the specimen consisted of mushy, partly soft, scaly grayish-white and partly indurated brownish fragments of tissue. Histologic examination revealed lamellated, keratinized material with areas of many polymorphonuclear leukocytes and a few lymphocytes and plasma cells. Centrally the lamellae were thrown into folds around a core of hyalinized keratin containing some calcium deposits. No cholesterol clefts were seen. (Fig. 1.)

Roentgenography revealed only slight haziness of the air cells of the left mastoid, and with this finding, together with the relatively excellent hearing (25 db. loss in the speech frequencies) a diagnosis of external cholesteatoma was made.

Conservative therapy was attempted. Ear drops were prescribed; cholesteatoma material was removed on two occasions; wicks were inserted into the canal in an effort to prevent stenosis, but it became evident that nothing short of a modified radical mastoidectomy would effect a cure.

On May 10, 1957, under general anesthesia, an endaural incision was made and a modified radical mastoidectomy was performed. The mastoid cavity was free of exudate and granulation tissue. The ossicular chain was intact and free of granulations. The bony external canal was enlarged from pressure necrosis. The bone was very thin superiorly, and the bridge was completely missing. The tympanic membrane was atrophic. The skin of the canal was largely replaced with granulation tissue, especially in the posterosuperior portion (Fig. 2), but a small flap was cut to cover the stapes and the head of the malleus.

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From the Otolaryngology Service, Department of Surgery, Tokyo U. S. Army Hospital. Present address: William Beaumont Army Hospital, El Paso, Texas.

Fig. 1.—In the upper left portion of this section is seen the central core of hyalinized keratin showing calcium deposits (rounded dark-staining areas). The remainder of the section shows lamellae of keratinized material. Near the center, the lamellae, which once were located peripherally, are thrown into folds. No cholesterol clefts were found in any of the sections. (Hematoxylin and eosin stain; × 75.)

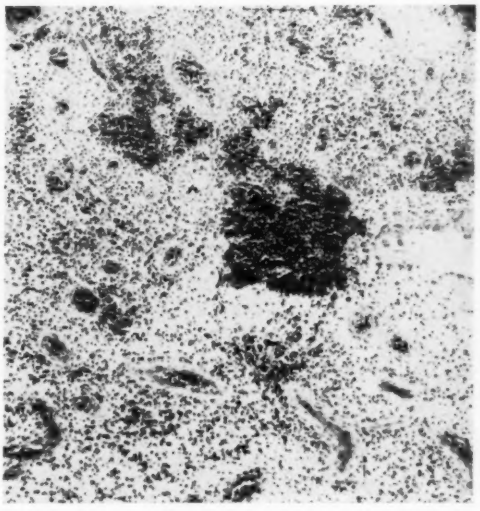

Fig. 2.—Granulation tissue, just beneath the epithelium of the canal skin. Note the numerous proliferating capillaries, edema, and cellular infiltrate, which on higher magnification was shown to consist of lymphocytes, plasma cells, macrophages, and rare polymorphonuclear leukocytes. Some of the sections showed hemorrhage and some showed strands of fibrous tissue and foreign-body giant cells. (Hematoxylin and eosin stain; × 75.)

Convalescence was uneventful. The skin flap that was laid over the incus and head of the malleus, however, atrophied. In spite of this, hearing had improved to the 17 db. level. It was planned to place a skin graft over the incus and head of the malleus; however, on July 9, it was observed that a membrane was growing outward from the edge of the tympanic membrane, downward from the skin flap that had been laid superiorly, and upward from the skin flap that had been laid inferiorly. This membrane continued to grow, closing in from the periphery and was nearly closed by Aug. 1. The small unclosed area was covered with a crust. It was hoped that the mastoid cavity would fill in with granulation tissue and that the final effect would be that of a large external auditory meatus. By Nov. 15, however, the crust fell off, bringing the mastoid cavity into view. The cavity was dry; the tympanic membrane was atrophic, wrinkled inferiorly, and flattened. Hearing remained at the 15 db. level.

Comment

It is interesting to note that, although this patient consulted a physician initially because of earache and was given local treatment, he never consulted a physician during the time when his only symptom was deafness. At the time of his physical examina-

tion for induction into the Army, he did call attention to his deafness, but according to the record, no abnormality was found in either ear and the whispered voice was heard at 15 ft. bilaterally.

Etiology.—For many years it was doubted that a cholesteatoma of the external auditory canal could occur without the simultaneous presence of a similar process in the middle ear,[8] and as late as 1953 one author was of this opinion.[10] This seems illogical.

Most writers acknowledge that protracted hyperemia of the skin of the external ear canal, leading to a chronic desquamative process, is the underlying condition which leads to external cholesteatoma. The hyperemia may have been started by otitis media, furunculosis, or inflammation of adjacent structures.[9] Dry eczema,[11] localized periostitis,[8] and seborrhea[12] have been mentioned as causative factors in some cases.

Therapy.—Treatment consists of mechanical removal of the mass. If this is done early, before the external auditory meatus is completely filled, erosion of the bony ear canal will be avoided in most cases. Periodic removal may be necessary.[5,9] Aqueous irrigations may be helpful in the removal of loose fragments, but this leads to swelling of any remaining material and should therefore be followed by the instillation of alcohol. After cleansing has been completed, any granulations should be treated until healed.[9] In the case reported herein, so much of the canal skin was replaced with granulation tissue that modified radical mastoidectomy was performed in order to prevent stenosis of the external auditory meatus.

Nomenclature.—The clinical term "cholesteatoma" has been used by otolaryngologists for many years. It should not be confused with "true cholesteatoma" of bone, which develops from congenitally displaced epidermal cells. Eggston and Wolff[13] stated that the aural cholesteatoma is more accurately termed "epidermoid cyst," and Fowler[14] referred to it as an "inclusion cyst."

Summary

Protracted hyperemia of the skin of the external ear canal, leading to a chronic desquamative process, is the underlying condition which leads to external cholesteatoma.

The case herein presented was cured by modified radical mastoidectomy, and hearing was markedly improved.

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Clinical Notes, New Instruments and Techniques

Inhalation Analgesia in the Office Practice of Otolaryngology

IRVIN FELDMAN, M.D., Washington, D. C.

From the Department of Otolaryngology, Georgetown University School of Medicine.

There is a need in the office practice of otolaryngology for a safe and simple inhalation analgesic for minor surgical procedures. Such an analgesic is particularly adaptable to myringotomies and incisions of furuncles of the external canal, short but painful procedures.

Trichloroethylene (Trilene *) appears to be ideal for this purpose. The induction is rapid; no complicated apparatus is needed; no premedication is required; fasting is not necessary since nausea and vomiting do not occur, and recovery is rapid without side-effects. Virtually all of the trichloroethylene is removed from the body within a minute or so after the last inhalation.

Trichloroethylene is a stable, clear liquid, which is tinted blue by the manufacturer. Its odor is fruity and not unpleasant. Most important, it is nonexplosive and nonflammable when administered with air at room temperatures. The drug is relatively nonirritating to the respiratory tract, and it does not affect blood pressure or increase bleeding.

Technique of Administration

Arbitrarily, trichloroethylene was not administered to children under 2 years of age. Below this age, children can be easily managed, with proper holding, without anesthesia for such short procedures. Above this age, they become more difficult, and pain and emotional problems become more significant.

At the onset, an attempt was made to use an inhaler, but, particularly in children, the approach of the mask, even after explanation, caused a great deal of excitement and struggle. At the present time, a pad of gauze is saturated with the analgesic and squeezed until all excess liquid is removed. (Trichloroethylene is irritating to the skin, and, if allowed to drop on the face, will cause transient irritation and burning.) A nurse sits at the head of the patient so that at the proper moment she may turn the head to one or both sides for quick myringotomy, etc.

Submitted for publication Nov. 21, 1957.

* Trilene is manufactured by Ayerst Laboratories, 22 E. 40th St., New York 16.

Prior to starting, the administration is explained to the patient, if he is old enough to understand. The pad is held about 3 to 4 in. from the nose, and the patient is instructed to count slowly. As a rule, by the time he has counted to between 25 and 50, the numbers become confused and slurred. At this stage, or when he stops counting, the patient is ready, and the operative procedure is accomplished. There may be slight motion or disturbance during the procedure.

If an intramuscular injection such as penicillin is to be given, this should be ready in advance, and there is time to administer this without pain.

After the analgesia, the patient may open his eyes within seconds or fall into a natural sleep from which he can be easily aroused. No nausea or vomiting is encountered. There is a period of amnesia for the time the patient was under the influence of trichloroethylene. Patients have stated that during the procedure they noted some sensation of dizziness and black spiraling circles. This was followed by a dream, not unpleasant, the details of which were not recalled.

Adults and older children are instructed to hold the gauze with the trichloroethylene and to breathe on count. At the point where analgesia occurs, the hands will drop, and the operative procedure can be performed. With younger children, and those not cooperative, the gauze is held by the surgeon while the patient counts or cries. The patient is ready when the crying stops. These patients are usually held by the hands and feet, but restraints have never been necessary. Because of increased respiration during crying, the period before analgesia occurs is shorter as a rule. Occasionally, the child will hold his breath just after starting. The gauze is withdrawn until respiration recurs and then continued as previously described.

Contraindications

Trichloroethylene has a wide margin of safety when used only for analgesia. Disturbances of cardiac rhythm occur when the concentration of trichloroethylene is increased to the level required for surgical anesthesia. However, analgesia will not be exceeded if the drug is self-administered or removed instantly when consciousness is lost or

tachypnea occurs. Tachycardia, bradycardia, or occasional hypotension may occur. Cardiac and respiratory arrest have been reported with heavy concentrations.

According to some authors, liver function has been temporarily impaired by high concentration, and its use is not recommended in patients with liver disease, dehydration, or cardiovascular diseases. Epinephrine is contraindicated with trichloroethylene for fear of sensitizing the heart. Trichloroethylene is not used in a closed system with soda lime, as toxic products (dichloroacetylene) may be formed which are capable of causing cranial nerve palsies.

Summary

On the basis of three years of personal experience, trichloroethylene (Trilene) is recommended for office use in otolaryngology for short but painful operations, such as myringotomy and surgical treatment of external otitis. By the method described, it is possible to get quick, safe analgesia which by its simplicity greatly facilitates the performance of these minor surgical procedures.

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Stapes Mobilization and Tympanoplasty Instruments

1. The Pencil Burr

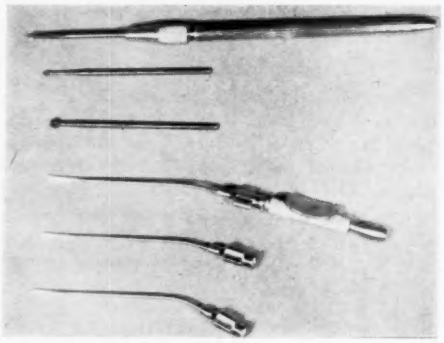
2. Cotton-Tipped Suction Needles

J. BROWN FARRIOR, M.D., Tampa, Fla.

These two instruments, the pencil burr and cotton-tipped suction needles (Figure), have been a great help to me in obtaining adequate exposure of the footplate and a dry field in stapes mobilization surgery. In tympanoplasties the moistened cotton-tipped suction needles make delicate dissectors for elevating the squamous epithelium from the drum remnants and canal wall.

The pencil burr consists of a small burr placed in a universal chuck handle as used for a laryngeal mirror (or formerly the lead burr). By rotating this burr between the fingers it acts as a rotating curette to remove the bone of the canal and expedite the exposure of the footplate. The 1- or 1.5-mm.-long shanked cutting burr is far more rapid than the curette but leaves some bone chips. The 1 or 1.5 mm. polishing burr is smoother and leaves no bone chips; it does, however, become clogged and requires changing.

The moistened cotton-tipped suction needles will remove blood without trauma to the delicate flap or mucous membrane of the middle ear. They are of value in elevating the flap. V. Mueller and Storz have made these needles by triangulating the ends of the 17 gauge, 20 gauge, and 22 gauge suction needles. We have made some of our own by roughening the end of existing suction needles with a file. During operations about six of each size are kept prepared for frequent changes.



The pencil or hand burr aids in exposure of the stapedial footplate. A small whisp of cotton wrapped around a suction needle creates a delicate dissector and atraumatic suction for tympanoplasty and stapes surgery.

Submitted for publication Aug. 12, 1957.

Bay Shore Blvd. at Hyde Park Ave.

A New Tongue Depressor

ERWIN H. ROCK, M.D., Yonkers, N. Y.

The tongue depressor shown here was designed primarily for use during adenotonsillectomy under general anesthesia, but can be utilized where a tongue depressor otherwise is needed.

As can be seen from the figure, the tip of the instrument is curved. This was done so as to be able to lift the base of the tongue upward and forward. The larger blade of the instrument measures 12 cm. in length, and the width is 1.8 cm.; the smaller blade is 10 cm. long and 1.5 cm. wide.

Thus, the instrument can be used for adults and children alike because of the two different dimensions on the one instrument, the smaller blade serving as a handle while the larger blade is used, and vice versa.

It has been such a common experience in the past for an intern or for me to obstruct the patient's airway with the tongue depressor during general anesthesia. This is primarily due to pressure forcing the base of the tongue with the epiglottis downward and backward, with the resultant airway obstruction. The anesthetists constantly remark about this practice, and rightfully so. True, this practice can be prevented with any tongue depressor, provided the base of the tongue is lifted upward and forward. This tongue depressor was specifically designed to allow tongue elevation or protrusion from the base as a more natural procedure.

The blades were so constructed that it has no holes in it for the tongue to flare through and is narrow enough to allow good exposure. It serves as a tongue elevator rather than a tongue depressor. The curvature at the tip of the blades takes the shape of the curvature of the base of the tongue. Likewise, the curved tip aids in exposing the inferior aspect of the tonsillar fossa. The serrated ends on the bottom of each blade are so arranged that they run obliquely, tapering toward the handle. This allows the tongue to be grasped more firmly and without undue trauma. Finally, these serrations aid in grasping the blade when it is used as a handle.

This tongue depressor is being manufactured by The Lawton Company, 425 Fourth Ave., New York 16.

Submitted for publication Sept. 5, 1957.

Instructor in Surgery (Head and Neck Division), Albert Einstein College of Medicine, New York.

239 Park Ave.

Abstracts from Current Literature

Nose

SINUSITIS IN INFANCY. C. BEILLE, J. franç. oto-rhino-laryng. 6:3055 (March-April) 1957.

This presentation describes the various types of sinusitis that may occur in infancy and young children. Among the acute sinus attacks, the acute ethmoiditis is the commonest. This very frequently is first recognized by an appearance of an orbital abscess, usually in association with an acute upper respiratory infection. The acute frontal sinusitis occurs usually in the older children mainly between the ages of 5 and 10 years and is a relatively rare occurrence. The acute maxillary sinusitis is relatively frequent after the age of 4 years, and the frequency increases with the increase of age. The acute sphenoidal sinusitis does not appear before the age of 10 years. Chronic sinusitis usually involves either the ethmoids or the maxillary sinuses. They may be apparent or latent. In the latent form they may present polypi either of ethmoidal or of maxillary origin.

The complications are rather diverse. They may involve either the orbit, the endocranium, the respiratory tract, or the organism as a whole. The orbital complications may be either serous or suppurative. They may involve the orbit proper (iritis, iridochoroiditis, and dacryocystitis); the optic nerve may also be involved. The intracranial complications may occur rather frequently, such as an acute or chronic osteomyelitis, thrombophlebitis of the venous sinuses, meningitis, and encephalitis. Respiratory complications may involve any portion of the respiratory tract. Among the general complications may be articular rheumatism, Still's disease, renal complications, and many other complications due to either toxemia or bacteremia.

Various methods of diagnosis and differential diagnosis are discussed. Finally, treatment is subdivided into (1) medical and (2) surgical measures. The medical measures deal with decongestion, drainage, and disinfection. They include the antibiotics, aerosol treatments, displacements, and even maxillary antral punctures. Stress is also placed on vaccine therapy, change of climate, and many other measures, such as vitamins, endocrine, thyroid, and physical therapy, depending on the type and persistence of the infection. The surgical measures include simple measures such as adenoidectomy, resection of the anterior tip of the middle turbinate, and antral punctures, and in the stubborn cases major surgical procedures such as the Caldwell-Luc operation or the operation of DeLima and trephining of the frontal sinus. Where allergy is suspected desensitization is rather difficult; however, the antihistaminics, the cortisones, and the sympathicotonic or parasympathicotonic drugs may act on the neurovegetative system.

PERSKY, Philadelphia.

DECORTICATION OF THE NOSE AND ITS VALUE IN COSMETIC SURGERY. A. SERCER, Rev. laryng. 18:161 (March-April) 1957.

The author employs a high transcolumellar incision to expose nasal structures. The skin is freely elevated over the cartilaginous and bony portions of the nose. This exposure permits a direct view for all procedures that may be indicated. The method is a slight variation of that proposed by Rethi. At the conclusion of the operation, the columella is replaced and a few small sutures are inserted. Several weeks later there is only a very small, scarcely perceptible scar.

PERSKY, Philadelphia.

Pharynx

DIRECT ADENOIDECTOMY. PAUL GUGGENHEIM, Eye, Ear, Nose & Throat Month. 35:653 (Oct.) 1956.

The goal of every adenoidectomy is a clean central vault. Direct inspection will quite frequently reveal tissue high in the vault which is difficult or impossible to detect with the most

sensitive finger. Tissue high in the vault may be removed with a very small adenotome, by avulsion with a clamp or forceps, or by the judicious use of a curet. Although the lateral lymphoid curtains do not cause postnasal obstruction even when hypertrophied, they are responsible for many of the poor results following adenoidectomy. They may be resected with an adenoid punch or sharp dissection with a small right-angled scissors. Under direct visualization, the tissue of Rosenmuller's fossae may be avulsed with an Allis forceps or resected with a biting forceps.

Considering the incidence of deafness secondary to otitis media in childhood, adequate adenoid surgery presents a larger statistical surgical problem than fenestration or stapedial mobilization. Tonsillectomy and adenoidectomy is a major procedure and should be performed by a qualified otolaryngologist equipped to perform a demanding surgical procedure.

ABSTRACTOR'S COMMENT: Any article stressing the importance of careful adenoid surgery deserves careful reading. Not only does the soft palate serve as a curtain to obscure surgical technique, but as a curtain it also hides incomplete results. Too often the adenoidectomy is a quickly performed postscript following tonsillectomy. Direct visualization during adenoidectomy can have a sobering influence on one who has thought that his finger was all-dependable in detecting adenoid tissue! The role of direct visualization in adenoid surgery should be considered akin to the role of the endaural surgical approach in surgery of the temporal bone.

JENNES, Waterbury, Conn.

THIRTY YEARS' EXPERIENCE WITH RADIATION THERAPY OF PHARYNGEAL LYMPHOID TISSUE. JOHN H. VAUGHAN, Eye, Ear, Nose & Throat Month. 35:787 (Dec.) 1956.

The author has used x-ray therapy in more than 6000 patients since 1926. Treatment is given weekly for five weeks. The dosage is 200 kv. at 15 ma., screened with 0.5 mm. of copper plus 1 mm. of aluminum. At each treatment, between 60 r and 100 r is given to each side of the neck, depending on the patient's age and the thickness of the neck. The portal used is large enough to cover Waldeyer's ring. Radiation therapy has been used in diseases of the pharyngeal lymphoid tissue either because of definite contraindications for operative intervention, in those patients who have refused surgery, or in patients in whom this form of therapy was the primary choice.

Of the 6000 patients so treated 25% had had previous tonsillectomy. The author feels that a favorable clinical result was obtained in approximately 90% of the patients treated, whether or not surgery had been performed. Favorable results usually occur two to three months after irradiation. It is rarely necessary to give a second series of treatments.

JENNES, Waterbury, Conn.

JUVENILE NASOPHARYNGEAL FIBROMA. E. G. GILL, J. A. THURMOND and C. E. LEBLANC, Eye, Ear, Nose & Throat Month. 36:32 (Jan.) 1957.

Juvenile fibroma of the nasopharynx is generally encountered in males between 6 and 20 years of age. Diagnosis is made by a history of progressive nasal obstruction with recurrent or persistent epistaxis in the presence of a large firm palpable mass attached near the choanae in the nasopharynx. Biopsy should be performed only in the operating room with adequate preparations for the control of severe hemorrhage.

Surgical removal is the usual method of treatment. This may often be difficult owing to the size and location of the tumor and the involvement of contiguous structures. Ligation of the external carotid artery preceding operation has been recommended to decrease the amount of bleeding. Since the majority of these tumors are radiosensitive, irradiation has been advocated as a treatment. Irradiation, which may be either external or internal, can reduce the size and vascularity of the tumor. Juvenile nasopharyngeal fibromata regress in size after puberty, and it is therefore often advocated that treatment be postponed. This delay, however, is not advisable because of potential damage to adjacent structures and the possibility of severe hemorrhage. A case report is presented of a 16-year-old boy with a juvenile nasopharyngeal fibroma which was treated by uneventful excision.

JENNES, Waterbury, Conn.

Miscellaneous

CORRECTION OF PROGNATHISM. IRVING B. GOLDMAN, Eye, Ear, Nose & Throat Month. 35:648 (Oct.) 1956.

Prognathism may be either hereditary or developmental. Preoperative examination should include intraoral and extraoral roentgenography and cephalometric study, profile and full photographs, facial masks, and impressions of the upper and lower teeth, from which casts are made. Orthodontic treatment before surgical intervention gives maximum intermaxillary fixation, and thereby improved postoperative occlusion.

There are two basic groups of prognathism: one, in which the entire mandibular arch is wider as well as protrusive to the maxillary arch; and two, where there is a good molar occlusion and relationship and the prognathism is limited to protrusion of the anterior part of the mandibular arch.

In the first group, osteotomy is the procedure of choice, and this is accomplished by a transverse or oblique division of the ascending ramus midway between the sigmoid notch and the mandibular sulcus, with shifting of the body posteriorly as a unit. In the second group, ostectomy is the procedure of choice, with removal of a section of the mandible on one or both sides of the molar or bicuspid area and repositioning the anterior section. This procedure may be in one or two stages, and the approach may be either intraoral or extraoral, or a combination of both. Several techniques for the surgical correction of prognathism are described in detail.

JENNES, Waterbury, Conn.

OBSTRUCTIVE LESIONS OF THE SALIVARY GLANDS. J. A. THURMOND, Eye, Ear, Nose & Throat Month. 36:29 (Jan.) 1957.

Obstructive lesions of the salivary gland are classified as follows: 1. Calculosis (sialolithiasis). 2. Stenosis. 3. Allergy. 4. Compression due to adjacent structures and extrinsic tumors. The order of frequency of the occurrence of salivary calculi is the following: Wharton's duct, the submaxillary gland, the parotid gland, and Stensen's duct. Acute obstruction of a duct is characterized by severe pain with swelling of the gland. Intermittent irritation, discomfort, and swelling of the gland usually indicate a calculus located in the substance of the gland. Diagnosis is confirmed by either palpation of the stone or by x-ray demonstration of its presence, with or without contrast media. The treatment is surgical removal of the calculus after the inflammatory reaction has been controlled by antibiotic or chemotherapeutic treatment. In the case of multiple stones or a large stone within the gland, or an associated chronic inflammation, the entire gland may have to be excised.

Stenosis or stricture of the salivary ducts causes symptoms similar to those of calculi. Diagnosis is made by probing the duct and by x-ray studies following injection of radioopaque substances into the duct. Occasionally it is necessary to make an artificial fistula into the mouth by-passing the stenosis. In those cases which are due to an intrinsic tumor, the gland and tumor must be excised.

Certain obscure obstructions may be due to edema of the duct walls caused by an acute allergic reaction. The diagnosis is made by exclusion and by finding an increased number of eosinophiles in a stained smear made of secretions from the salivary duct. Treatment follows usual allergic lines. When obstruction is due to pressure on a duct from adjacent structures, the treatment is removal of the extrinsic cyst or tumor.

Two case reports are presented, one of a calculus within the submaxillary gland treated by excision of the gland, the other of a stricture of Stensen's duct treated by dilatation.

JENNES, Waterbury, Conn.

SPEECH OF CHILDREN BEFORE AND AFTER REMOVAL OF TONSILS AND ADENOIDS. MARGARET C. L. GREENE, J. Speech & Hearing Disorders 22:361 (Sept.) 1957.

The speech of 377 children undergoing adenoidectomy and tonsillectomy was examined before surgery. There was a high incidence of articulatory defects in this group. Abnormal tongue postures were present in 5.6%; lisping in 11 cases; insufficient nasal resonance in a number of cases; defective articulation in 5.7%, out of those who underwent both operations, and in one of the 30 who underwent adenoidectomy alone. Enlarged tonsils may contribute

to difficulty in articulation. The speech of 158 of these children was examined after operation. Nasal speech occurred in 7.2% of cases following removal of tonsils and adenoids, and in 13.3% of cases following adenoidectomy. Recovery of normal speech was rapid except in the cases of four children with emotional problems. Hearing improved in all cases of catarrhal deafness after surgery.

PALMER, Wichita, Kan.

A POINT OF VIEW ABOUT 'STUTTERING.' DEAN E. WILLIAMS, J. Speech & Hearing Disorders *22*:390 (Sept.) 1957.

The speech behavior patterns usually associated with stuttering vary from person to person and from time to time with any given person, in many ways. The meanings the speaker gives to certain "feelings" and emotional states tend to vary. Such variability should prompt the clinician to discontinue talking about stuttering as if it were a vague, undefinable entity. Instead, the clinician should learn to deal with each subject by discussing, on a descriptive level, the speech behavior patterns as they vary from those involved in normal speech.

PALMER, Wichita, Kan.

SUCCESSFUL TREATMENT OF APHTHOUS STOMATITIS. MARTIN H. ZWERLING, New York J. Med. 57:2236 (July 1) 1957.

In August, 1955, Zwerling published his first article on Clorpactin in The Arch. OLR (62:157). He now reports his experiences with this chemical in aphthous stomatitis in 200 patients—100 in each series. In the first 100 (Clorpactin WCS-60 and WCS-90, Guardian Chemical Co., Long Island City), a 0.2% solution was made with tepid water, and used as a mouth wash and gargle every three hours. Occasionally some of the solution was swallowed, but no toxic or irritating symptoms were reported. Results: Of 100 cases of aphthous stomatitis,[91] showed "marked improvement within eight hours." However, where lesions occurred in the larynx, improvement was much slower, and increasing the dose of the remedy seemed not to change conditions greatly.

A second 100 patients were treated with WCS-90, and 98 "responded within eight hours." Two who had involvement of the larynx were refractory, but improved with increased dosage.

Clorpactin is basically hypochlorous acid. Good results have been reported from the Department of Urology at Presbyterian Hospital, New York; also by a dentist (T. M. De Stefano) before the New York State Dental Society. Zwerling says: "Tests show it to be a powerful virucidal agent without significant toxic or irritating effects."

ABSTRACTER'S COMMENT: The author is rather too casual in the use of the word "response." We assume that he means the patients were better, but where is the control? What laboratory work was done? If this agent is nearly 100% perfect in action the whole world needs to know about it. . . . Let's hope!

I. W. VOORHEES, New York.

ALLERGENS OF IMPORTANCE EASILY OVERLOOKED IN THE ETIOLOGY OF ALLERGIC DISORDERS. HAROLD GELFAND, New York J. Med. 57:2260 (July 1) 1957.

Gelfand works in the Department of Allergy in Gouverneur Hospital, New York. His contribution is part of the "Fundamentals of Modern Allergy" under the editorship of Samuel J. Prigal. The author stresses the unusual forms of allergy which may be by-passed because they are so subtle. Table 1 gives nonfood sources of gum allergens, and the list runs from adhesive plaster and artificial flowers to varnishes and water colors. Table 2 gives the food sources of gum allergens, and mentions karaya gum, gum arabic, and tragacanth. This list names salad dressings, mustard, charlotte russe, packaged cheeses, tooth paste, and chewing gum. In summary, we need to watch out for (1) animal emanations, insect dust, and odors and fumes; (2) vegetable gums by inhalation and contact; (3) certain chemicals used in printing, dyeing, and cosmetics; also such drugs as aspirin, quinine, and the barbiturates; (4) contaminants in milk and foods which are sprayed in the process of growing; (5) common and miscellaneous foods, coffee, mushrooms, buckwheat, cereal grains, eggs; (6) food adjuvants such as spices and condiments; (7) agents for infusion purposes, "plasma expanders", etc.

ABSTRACTER'S COMMENT: It seems that somebody somewhere under the sun is hypersensitive to any and all objects on earth. We have, therefore, the gigantic task of finding the enemy for each individual case. Recently a case came to my notice which seemed rightfully diagnosed as Ménière's with all the characteristic symptoms. The Rosen operation was advised. The patient demurred. An astute allergist inquired minutely into the patient's diet. His recommendations were followed. Result: no more dizziness or other symptoms for three months past. A patient who could not get out of bed without falling now visits the metropolitan stores alone! Marvelous? We need even a better word!

I. W. VOORHEES, New York.

FACIAL PAIN, ITS DIFFERENTIAL DIAGNOSIS AND TREATMENT. DOGAN M. PARESE, New York J. Med. 57:2807 (Sept. 1) 1957.

Parese works in the Roswell Park Memorial Institute in Buffalo. His experience covers "a great number of patients with facial pain in the special pain clinic." Diagnosis is of first importance, and so a complete evaluation of the entire patient is paramount. Classification is set up as follows: (1) inflammation; (2) trauma; (3) malignancy; (4) anomalies, and (5) neuralgias. Under (1) we observe dental, sinal, C. N. S. (central nervous system), miscellaneous infections (erysipelas, arteritis, etc.). Trauma from fractures of the mandible, from prosthetic appliances, and Costen's syndrome are mentioned. Malignancy in the soft or bony parts of the head, C. N. S. or cervical spine can be painful. Anomalies in blood vessels, teeth, jaws, etc., may be basic for pain. The neuralgias are "typical" or "atypical." An incisive study of laboratory and x-ray findings may be helpful, but is not sufficient per se. The meningitides, Gradenigo's syndrome with partial paralysis of the sixth nerve, and petrous bone necrosis, must be thought of. Fractures of the base of the skull will probably be recognized as potent causes of pain. Costen's syndrome deals with jaw-joint pain, spontaneous or elicited on pressure . . . (Look out for aural furunculosis!) Tumors of the nasal sinuses give a variety of symptoms which may confuse the examiner. Sensory nerve defects, e. g., decreased corneal reflex, lack of pin-prick response, etc., will be elicited during the neurological evaluation. Owing to the wide distribution of the fifth cranial, its branches must be traced thoroughly.

Neuralgia, typical or otherwise, supposedly signifies pain without inflammation. Trigeminal type may be due to arteriosclerosis of the gasserian ganglion (*Schaltenbrand*). It is often spoken of as migraine, but this adds nothing to the etiology. Pain in the eyeball and upper teeth is to be expected. Herpes sometimes follows the course of any branch of the trigeminal nerve. The author mentions geniculate neuralgia, tympanic nerve, greater superficial petrosal involvement, neuralgia of the vagus, and auriculotemporal types. Glossopharyngeal nerve pain is rare but easily identified by location along the side of the tongue and inferior teeth. . . . An injection of a local anesthetic may give immediate relief, permanency of which cannot be foretold. . . . Treatment must be based on accurate diagnosis. Dental pain may not always be due to "bad teeth." Occlusal anomalies will be easily found and can often be readily relieved, but time may be prolonged. Vitamins, especially the touted B_{12}, have been disappointing. The author speaks of Stilbamidine in tic douloureux but there may be undesirable side effects (paresthesias, formication) and "a greater degree of discomfort than before the use of the drug." Several paragraphs are given to "alcohol block," which may be useful—and dangerous (keratitis, loss of sight). Avulsion is mentioned but not stressed. Neurosurgical procedures may be indicated at times and can be simple or involve "deep invasion" of major nerve centers. "Sympathectomies are not successful in more than a small number of cases."

ABSTRACTER'S COMMENT: This is a thoughtful study of a subject which may be most difficult and unrewarding from a therapeutic standpoint. These sufferers will undergo any measure which offers a semblance of relief, but after going through it without much benefit they become resentful and out of patience with their medical manager. Block anesthesia of whatever kind offers direct, fairly simple technical procedures. They should be tried first before major surgery is undertaken. There seems to be less of the latter (removal of the gasserain ganglion) than was in order a quarter century ago. The author presents three diagrams and 37 references to the literature in support of his study. He is to be commended for his effort.

I. W. VOORHEES, New York.

ETIOLOGY AND SYMPTOMATOLOGY OF TEMPOROMANDIBULAR JOINT DISTURBANCES. ARTHUR S. FREESE, New York J. Med. 57:2837 (Sept. 1) 1957.

Let no one be overdisturbed by the author's definition: "The temporomandibular joint is a ginglymodysarthrodial joint which is almost unique." He is in dead earnest about his thesis and handles it well. Subluxation of this joint has been a popular theme for theses over the years. It is now brought up to date by this sponsor. Causation is set down under three headings: (1) microtraumata; (2) overclosure; (3) muscular hypertension. . . . The anatomy of the joint is reviewed and symptomatology discussed. As to the latter we have (1) pain in the joint, constant or on motion; (2) swelling and tenderness; (3) clicking or grating sounds which may be subjective only, or "heard across the room"; (4) stiffness in the joint and muscles, with limited mobility; (5) pain in the adjacent regions—temple or masseter muscles, pterygoid, affecting the throat and tongue, digastric and geniohyoid. In osteoarthritis, the joint pain is constant giving the sufferer no peace. It is fortunately unilateral. X-ray examinations may be of no aid since one cannot get a true picture of the minor details. These may be of high importance as to malfunction, but are elusive. Impaired hearing, tinnitus, vertigo, and neuralgias are common symptoms. The dentist may be able to correct malocclusion.

ABSTRACTER'S COMMENT: The author offers little as to treatment. This is disappointing; for the patient has a right to expect relief. There is often a disturbance of the joint fluid (synovial)—too much, more often too little—hence the grating sound. Injections into the joint of various fluids (anesthetics et al.) sometimes bring relief. Ligaments and muscles may be too tight or too loose. What about "rheumatism" elsewhere in the body? This can be treated by "the newer remedies." . . . Open operation has been done to prevent or to overcome fixation. . . . The meniscus has been removed or replaced (stitched).

I. W. VOORHEES, New York.

COMPLICATIONS OF ANTIBACTERIAL THERAPY. GERARD EASTMAN, New York J. Med. 57:3119 (Oct. 1) 1957.

From the Department of Medicine, State University of New York Medical School in Syracuse, Eastman, working under two "grants," gives the results of his studies on a subject of great importance to every doctor of medicine, whatever his specialty. The enormous wave of enthusiasm welcoming antibacterial agents, as so often happens, has waned with experience. The author states that production by manufacturers "is measured in tonnage." This report aims to find the facts which make indiscriminate use of the antibiotics unwise. There have been some fatalities. Untoward manifestations are considered under three headings: (1) purely allergic symptoms and signs, such as rashes and urticaria; (2) toxic and metabolic effects, such as auditory (loss of both vestibular and cochlear functions) and negative nitrogen balance; (3) disturbances due to changes in otherwise normal bacterial flora in body cavities and tissues. Penicillin is the most widely used of all; reactions to it are chiefly allergic and occur in some 4% of all patients. This is true even for oral administration, but less so than by needle. Sensitization may be present, although the patient may never have been treated with penicillin. It may be latent in milk or poliomyelitis vaccine! Incidence following penicillin in oil is about 2.7%; for procaine penicillin 1.4%, and for aqueous solutions 1.2%. Penicillin lozenges can produce allergic symptoms. We need not look for a foreign protein in our preparations, for penicillin alone is quite capable of inducing all undesirable sequences.

As for eruptions, they may be quite simple or very severe, such as acute exfoliative dermatitis which may be fatal! Anaphylactic reactions are by far the most dangerous, and it is said that over one-third are fatal.

Intradermal skin testing preceding administration of penicillin is much in use, and does help considerably, but may not be entirely reliable. To overcome many objections, various types of penicillin have been brought out, such as the O, to circumvent unpleasantnesses, but as the author states, susceptibility to any one type penicillin means reaction to any other. Nature does not so discriminate. Antihistamines as antidotes are of questionable value. Severe reactions may come from too generous use of penicillin in body cavities during surgery.

Treatment of these allergic manifestations of penicillin must be symptomatic. In a sudden attack of asthmatic breathing, one may inject 0.1 to 0.5 cc. of epinephrine subcutaneously or intravenously. Oxygen should be at hand. Assuming that the penicillin has been injected into

an arm, one may well apply a proximal tourniquet to prevent too rapid absorption. In severe or prolonged undesirable instances, one may resort to adrenal cortical steroids. So much for penicillin.

Streptomycin and dihydrostreptomycin are more likely to prove toxic than allergic. Neurotoxic symptoms will likely appear if repeated or prolonged use of these two are deemed necessary. Sensitivity may show up in a week, or a month, or in six months. When either of the above is injected into joints, unpleasant conditions may be noted.

Para-aminosalicylic acid or "PAS" used in tuberculosis causes gastrointestinal upsets chiefly. These can be reduced or regulated by careful management. The good is likely to overcome the bad.

The tetracyclines, chloramphenicol and erythromycin, are broad-spectrum agents less likely to bring about allergic reactions than to cause gastrointestinal effects, the pathogenesis of which are not as yet understood, but shifts in bacterial flora may be partly accountable. It is well to use caution in treating any patient with chloramphenicol.

The sulfonamides seem relatively safe when used with discretion, a statement which goes for all drugs. A table (No. 2) from the researches of E. M. Yow shows the incidence of dermatitis, gastrointestinal disturbances (nausea, vomiting, and diarrhea) with certain blood effects (dyscrasias), leukopenia, and agranulocytosis; but all these were reversible after withdrawal. There were 1000 cases in the series.

Bacitracin and Polymixin-B have similar reactions, but have few allergic complications. There is, however, albuminuria and depression of tubular and glomerular functions. If these antibiotics are administered daily, a close watch must be kept on blood and urine. " . . . Reservation of its [their] use to situations where no other drug is effective" should be the rule.

Superinfections may be worsened by antibiotics since certain natural microorganisms of use to the body may be so attenuated that vicious germs entering the body have a free way. "Streptomycin is followed by superinfections in as many as 13% of cases; chloramphenicol in 15%, and the tetracyclines in 10% of cases." In summary, these drugs are not (any of them) entirely harmless. No drug will suppress all known bacteria. Occasionally, a situation may be made worse by their use.

ABSTRACTOR'S COMMENT: This is a splendid and authoritative résumé of our knowledge so far in the use of these valuable agents, but like a two-edged sword, they may cut for and against. Patients often regard antibiotics as harmless. Worse yet, they may cudgel a physician into use against his better judgment, or he may give excessive doses because his patient seems to be growing worse. To strike a nice balance calls for an intimate study of every patient who is undergoing such medication. The laboratory must be in constant touch with secretions and excretions. Blood and urine examinations daily will help greatly in estimating just how much or how little the given patient can withstand. Here is a "Doctors' Dilemma" which would have delighted the late G. B. S.

I. W. VOORHEES, New York.

CORTISONE IN OTORHINOLARYNGOLOGY. J. BONNEFOY, J. franç. oto-rhino-laryng. 6:677 (July-Aug.) 1957.

The corticosteroids have increased in their uses in the last years. With the newer preparations, the side-effects have been lessened, and a wider application of these drugs has occurred. In otolaryngology many indications for their use are found.

Locally, the cortisones have been used in the ear for external otitis, particularly during the acute state. The δ-cortisone, either the suspension, the aqueous solution, or the ointment, has given relief of symptoms within 24 to 48 hours. The author also found excellent results after a tympanoplasty where he filled the middle ear cavity with the solution. This helps prevent a recurrence of the adhesions in this cavity.

Intranasally, excellent results have been achieved by topical application, particularly in allergic rhinitis, with or without polyposis. However, intramaxillary instillation has not yielded startling results.

When cortisone is given internally there are spectacular results in selected conditions, such as allergic nasosinusitis, nasal polyposis, tubotympanic catarrh, or acute Eustachian occlusion on an allergic basis. Other conditions have responded to these drugs, such as acute corrosive

esophagitis, laryngeal edema, either allergic or secondary to radiation therapy, facial paralysis, and a number of infections involving the pharynx and tonsils.

The author concludes that this is a very important adjunct to the therapy of otorhinolaryngology.

PERSKY, Philadelphia.

News and Comment

ANNOUNCEMENTS

Third International Congress of Allergy.—The Third International Congress of Allergy, sponsored by the International Association of Allergology and the French Allergy Association, will be held in Paris, Oct. 19-26, 1958.

The program is as follows:

(*a*) Symposia on asthma and emphysema, immunology, recent clinical advances, biochemical aspects, autoimmune reactions, dermatology, and socioeconomic aspects. Participants are world authorities in special fields: Pasteur Vallery-Radot, Löffler, Grabar, Dixon, Chase, Coombs, Forsham, Sir Henry Dale, Halpern, Schild, Harrington, Ackroyd, Dausset, Zondek, Sulzberger, Jadassohn, and many others.

(*b*) Sectional papers, limited to 10 minutes, on any phase of allergy.

(*c*) Round table luncheon conferences of small groups on selected subjects, led by international authorities.

(*d*) Pre- or Postconvention tours.

(*e*) Social activities. A very enjoyable program has been arranged.

For all information regarding the program and papers to be presented please write to the following:

United States and Canada: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago, Ill., U. S. A.

Latin America: Dr. Jose M. Quintero Fossas, Paseo 313, Velado, Havana, Cuba.

Europe and other countries (and for Congress information and registration): Dr. Bernard N. Halpern, 197 Boulevard St. Germain, Paris 7, France.

Registration fees: Congressists $32. Relatives of Congressists $15.

Information on travel and pre- or postconvention tours may be obtained from Thos. Cook & Son.

Books

Fortschritte in der Hals-Nasen-Ohrenheilkunde (Advances in Oto-Rhino-Laryngology). Edited by L. Rüedi, M. D. Volume IV: Stapes Mobilization. Histological Atlas. By L. Rüedi and H. Spoendlin, Zurich: Clinical Report By F. Altmann, M.D., New York. Price, 39.50 Swiss francs. Pp. 180, with 104 plates. S. Karger, AG., Arnold Böcklinstrasse 25, Basel (American agent—Mr. Albert J. Phiebig, P. O. B. 352, White Plains, N. Y.), 1957.

We have here an excellent up-to-date report on the histology of the inner ear and the surgical procedures used in freeing the footplate of the stapes as compared with fenestration. French, German, and English texts make it possible for practically every otologist to appraise the latest utterance on chronic deafness of a nonneural nature. The histological data are based on 79 ears from 54 patients. While this enormous effort involves patient and extensive laboratory studies, the authors admit that not much value can come to the surgeon who is about to perform an operation for the relief of deafness. It cannot be otherwise. However, the important conclusion is that the pathology may involve not only the footplate of the stapes, but the cochlear capsule, the internal auditory canal, and the osseous labyrinth. Not only the oval window but also the round window may be sclerosed. In such case we are stymied, for there is no way of knowing before or even during operation whether we have merely a fixation which can be released surgically or whether the disease process has gone much beyond such localization. So much for that!

Dr. Franz Altmann came to the United States from Vienna many years ago and allied himself with the Columbia-Presbyterian Medical Center in New York City, where he is a Clinical Professor of Otolaryngology. He is an authority on every phase of the inner ear. We are likely to think of surgery for deafness as quite modern, following Sourdille of France and Lempert of New York, with many other workers in this field. Not so! Sixty years ago several otologists—Kessel (1875 and 1894), Mach (1870), Miot in France, and later Dench and Jack and Blake in the United States—concerned themselves with relief of fixation of the stapes. Some of the results were fairly good, too, but for some reason the enthusiasm died out for about 50 years. Then it came to life again. Fenestration was advanced by Sourdille of Nantes, France, and Holmgren of Sweden, among others, but it was not popularized in this country until Lempert attacked the problem and gave new life to the surgical enterprise. Previously three methods had been used: (1) to mobilize the stapes; (2) to extract the footplate; (3) to fuse the drum to the incus (tympanoplasty). Miot reported 200 cases with fair results, but there were infections, better hearing with insufferable tinnitus, or relapses, all of which seem to have discouraged these pioneers. Curiously enough the problems faced today differ little from those which existed from 1875 to 1900. The lapse of interest from 1900 to 1950 is remarkable in the light of present enthusiasm. Three reasons may be held responsible for this inertia: 1. Visualization was imperfect. 2. Infections were common (no chemotherapy). 3. Attention became directed more strenuously to the conquest of the "chronic running ear" and its surgical complications. Deafness was "just one of those things." In some respects it still is, but this book has to do with "Advances" . . . Altmann goes into minute detail of the technique worked out by Fowler, Jr., Chief of Staff and Professor of Otology in the Medical School of Columbia, and his associates. The results are, to be sure, far from perfect, but give cause for courage and persistence. Altmann summarizes as follows: "Nevertheless . . . the conclusion seems justified that the mobilization will, to a large but undetermined extent, replace the fenestration operation, even if it fulfills only a part of the promise it seems to hold."

This is a field for the young man who thinks that ENT is a "dead specialty." The exacting technique and nervous strain incident to working in a tiny field, where the services of an assistant are almost nil, can prove too great for "the older men."

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.
Place: Nova Scotian Hotel, Halifax, N. S. Time: June 9-11, 1958.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.
Meeting: Third International Congress of Allergology.
Place: Paris. Time: Oct. 19-26, 1958.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh International Congress of Bronchoesophagology.
Place: Kyoto, Japan. Time: Sept. 12-14, 1958.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Rio de Janeiro, Brazil. Time: Aug. 10-16, 1958.
President of Congress: Prof. R. David de Sanson.
Secretary of Congress: Dr. Walter Benevides, Caixa Postal 2838, Rio de Janeiro.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Alden Miller, 500 S. Lucas Ave., Los Angeles.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco. Time: June 23-27, 1958.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Erling W. Hansen, 90 S. Ninth St., Minneapolis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN BOARD OF OTOLARYNGOLOGY

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Walter Hoover, 605 Commonwealth Building, Boston.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Harry P. Schenk, 326 S. 19th St., Philadelphia 3.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: Fairmont Hotel, San Francisco. Time: May 19-20, 1958.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

SECTIONS:

Eastern.—Chairman: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Southern.—Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. Francis L. Lederer, 860 N. Lake Shore Drive, Chicago.
Western.—Chairman: Dr. Ben. R. Dysart, 960 E. Green St., Pasadena 1, Calif.

AMERICAN RHINOLOGIC SOCIETY

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, October, 1958.
Annual Meeting: Chicago, October, 1958.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: Fairmont Hotel, San Francisco. Time: May 17-18, 1958.
Place: The Homestead, Hot Springs, Va. Time: 1959.

AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, INC.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY

President: Dr. Irvin J. Fine, 505 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

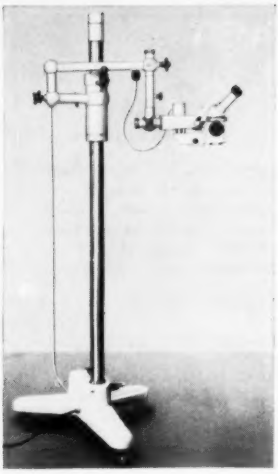
President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.
Place: Palmer House, Chicago. Time: Oct. 16-17, 1958.

OTOSCLEROSIS STUDY GROUP

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS

President: Capt. William C. Livingood, U. S. N. (MC).
Secretary-Treasurer: Lt. Col. Stanley H. Bear, USAF(MC), 3810th USAF Hospital, Maxwell AFB, Alabama.



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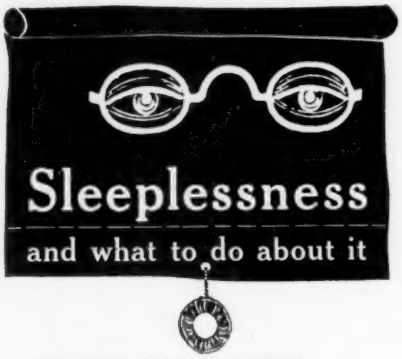

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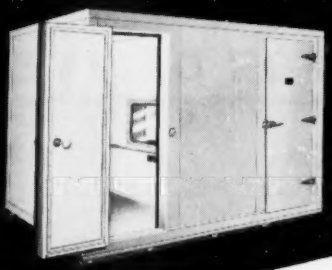

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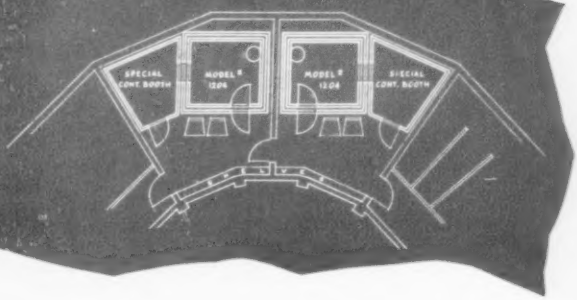

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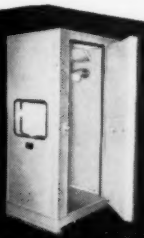

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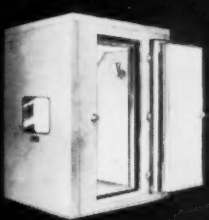

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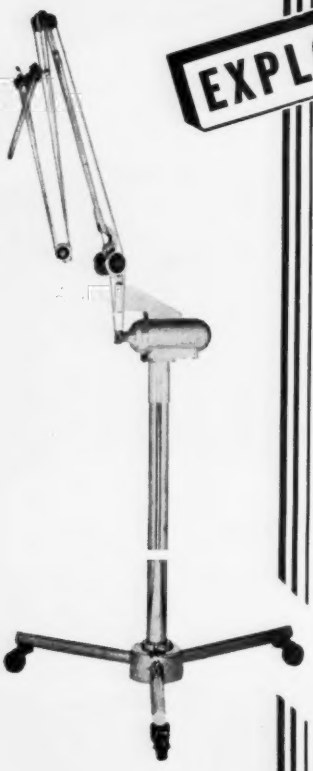

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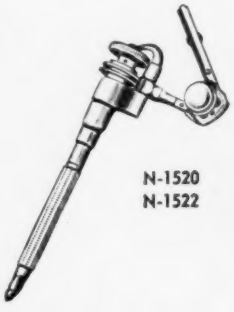

N-1520
N-1522

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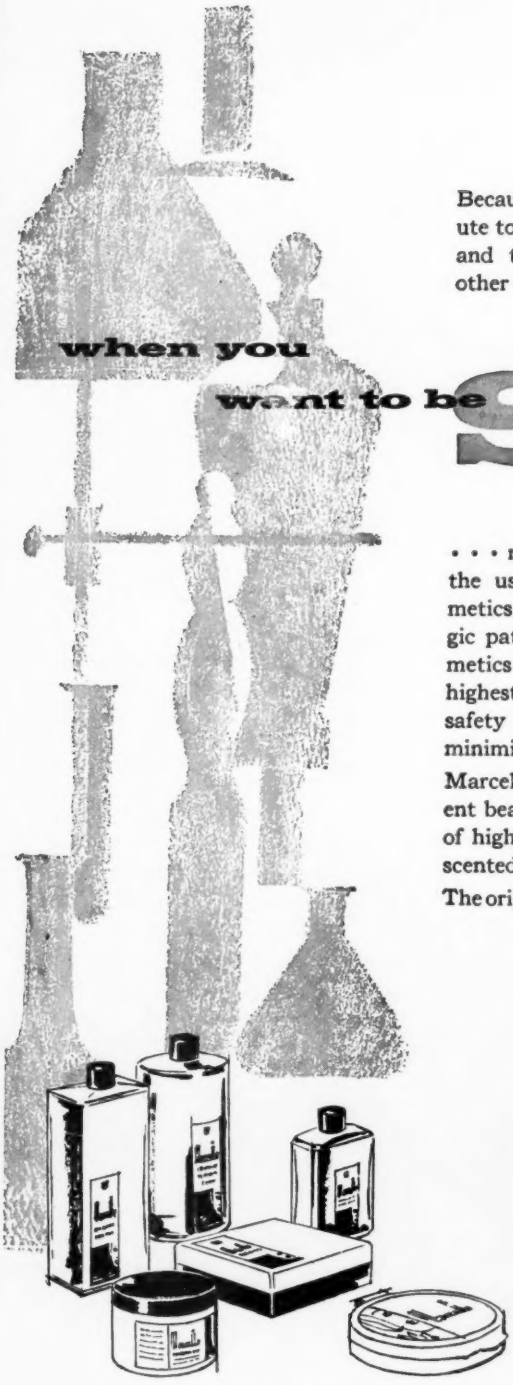

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